Tyson (Jas)

THE CAUSAL LESIONS

OF

PUERPERAL CONVULSIONS.

A PAPER READ BEFORE THE

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THE CAUSAL LESIONS OF PUERPERAL CONVULSIONS.

The writer would state at the outset that the time allowed for the preparation of this paper, had it included a much longer period, would have been altogether insufficient to have enabled him to base the conclusions arrived at upon personal observation. Cases of puerperal convulsions are fortunately not common, those which terminate fatally are less frequent, and *post-mortem* examinations still more rare. Of those which recover, very many occur under such circumstances that a previous clinical study is impossible. Thus, of five cases of puerperal eclampsia which have come under his observation in a practice of fifteen years, it so happened that none was seen until she was in the throes of labor. So that an examination of the urine in advance was impossible. All were primiparæ. In two of them, the subjects of which recovered, the convulsions did not occur until after the birth of the child, and did not reappear after a copious bleeding. They occurred very early in his practice, and no examination of the urine drawn by a catheter, and therefore separate from the lochial discharges, was made. They are therefore valueless. The third case, which again was not seen until convulsions set in, in what appeared to be the eighth month of pregnancy, occurred comparatively recently. The history developed the fact that the patient was enormously œdematous for some time before the convulsions occurred. The water drawn off by the catheter was highly albuminous, and contained granular and blood casts. She was not bled. Premature labor was induced, and she died about twenty-four hours after delivery. No *autopsy* was made, but the case was undoubtedly one of Bright's disease, and might be legitimately used in drawing conclusions.

In the fourth case of eclampsia, the urine was not examined until convulsions set in, but when examined immediately after the first convulsion, it was albuminous. In the first drawn, no tube-casts were found, but in the second and subsequent quantities, albumen, blood-disks, and tube-casts were present. These diminished gradually, and subsequently disappeared, the patient recovering completely. There was no œdema.

The specimens from the fifth case are now presented to the Society.

The contents of the larger four-ounce bottle, evidently bloody and highly albuminous, were drawn by a catheter after five convulsions had occurred, and *before* labor had set in. It represents twelve hours' secretion, and contains numerous highly granular casts, stained with hæmatin, blood-casts, epithelial casts, and blood-corpuscles.

The smaller bottle contains a portion of four ounces, drawn after she had been bled twelve ounces, and had received chloral, potassium bromide, a purgative dose of calomel, and an injection; had fallen into labor, and been rapidly delivered. Immediately after labor, she had received one-fourth of a grain of pilocarpin hypodermically, and fifteen minutes later was in a profuse perspiration and salivation, intelligence increasing momentarily. All this transpired in three hours after the first urine was drawn, and the four ounces, therefore, represent three hours' secretion. It is evidently lighter hued than the first specimen, but still contains considerable blood, is also highly albuminous, and contains large numbers of hæmatin-stained granular casts, blood-casts, and blood-disks. In both of the latter cases there was evidently Bright's disease.

This paper must consist, therefore, chiefly in an analysis of the work of others on the subject, and deductions from such examinations; and the only qualification which the writer may be said to possess for such a task is the interest he has hitherto shown in renal diseases, in a somewhat close study of their symptoms, and in the examination of urine derived from them.

In the collection of material upon this subject, it has seemed convenient to arrange it under one of the following heads:

I. Writers making the causal lesions of puerperal convulsions, in the main, identical with those of Bright's disease, in most cases that form known as parenchymatous nephritis (*syn.*, acute catarrhal or desquamatous nephritis, etc.), of which the typical example is the acute nephritis concurrent or sequel to scarlet fever.

II. Writers whose observations and belief tend to controvert the view that puerperal eclampsia is due to Bright's disease.

III. Writers who admit that puerperal eclampsia is sometimes due to Bright's disease, developed as a consequence of pregnancy, but who also believe that it occurs as often independently of such cause.

I. *Writers who consider the causal lesions of puerperal eclampsia to be, in the main, identical with those of Bright's disease.*

Cazeaux says that, with the exception of six or seven cases mentioned by MM. Depaul and Mascarel, the circumstance of the almost constant

presence of albumen in the urine of eclamptic women entirely escaped the notice of the older observers.

Rayer, in his *Traité des Maladies des Reins*, published in 1840, first announced that he had repeatedly observed his Nephrite albumeneuse in pregnant women. His pupil, Cohen, at his master's suggestion, wrote a thesis, in which he attributed puerperal albuminuria to nephritis. But although this relation seems to have been mentioned by Blackall and Tweedie in England,* Dr. John C. W. Lever is generally acknowledged to be the first in Great Britain, and, with the exceptions named, the first in the world, to point out the relation between albuminuria and puerperal convulsions. Dr. Lever reports in *Guy's Hospital Reports, Second Series*, 1842, fourteen cases, in ten of which the urine was examined: albumen was found present in greater or less quantity in nine cases. In the tenth case, which was fatal, and in which no albumen was found in the urine, the *post-mortem* examination revealed acute meningitis, with four or five drachms of milky fluid in the ventricles. The note describing the condition of the kidneys reads as follows: "The kidneys gave out much blood, but were not dark; their texture coarse and flabby." It is impossible to infer from this description whether they were diseased or not. In the remaining fatal case, in which the urine was highly albuminous before delivery, with a sp. gr. of 1010, but free from albumen after delivery, no *post-mortem* examination was permitted.

As to the cause of this condition, Dr. Lever was of the opinion "that the gravid condition of the uterus, by its pressure, prevents the return of the blood through the emulgent veins; and hence is the cause of the renal congestion, and the consequent albuminous condition of the urine." This opinion he considered supported by the fact "that the urine was found to be albuminous only in those women who were affected by or had premonitory symptoms of convulsions."

In 1846, Cohen and Delpeck published a memoir demonstrating the extreme frequency of albuminuria in pregnant women. The late Dr. Simpson, of Edinburgh, was one of the first to call attention to the relation of albuminuria to puerperal convulsions. In 1847 he detailed to the Edinburgh Obstetrical Society (*Edinburgh Monthly Med. Journal*, 1847, p. 288) "some cases illustrative of the effects of Bright's disease, as denoted by the appearance of albumen in the urine, under the action of heat and nitric acid," from which he drew the following conclusions:

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* See a valuable paper by Dr. Samuel C. Busey, on the "Artificial Induction of Premature Labor in Uræmia," read before the Clinico-Pathological Society of Washington, D.C.

1. Albuminuria, when present during the last periods of pregnancy, denotes a great and marked tendency to puerperal convulsions.

2. Albuminuria, in the pregnant and puerperal state, sometimes gives rise to other and more anomalous derangements of the nervous system without proceeding to convulsions; and Dr. S. had especially observed states of local paralysis and neuralgia in the extremities, functional lesions of sight (amaurosis, etc.), and hearing; hemiplegia and paraplegia more or less fully developed.

3. Œdema of the face and hands (going on occasionally to general anasarca) is one of the most frequent results of albuminuria in the pregnant female.

4. The presence of this œdema (3), or of any of the lesions of the nervous system (2) with or without the œdema, should always make us suspect albuminuria; and if our suspicions are verified by the state of the urine, we should diligently guard by antiphlogistic means, etc., against the supervention of puerperal convulsions.

5. Albuminuria and its effects (1, 2, 3) are far more common in first than in later labors, and these constitute a disease which in general disappears entirely after delivery. But Dr. S. had seen one case commencing with slight blindness, but no œdema, and ending gradually in hemiplegia, where the palsy partially remained after delivery, and after the disappearance of the albuminuria. In another, amaurosis came on with delivery, and had been present for six months when Dr. S. first saw her. She had no œdema or other symptom of albuminuria except the amaurosis; but on testing the urine it was highly albuminous.

6. Albuminuria with convulsions, etc., occurring in any labor later than the first, generally results from fixed granular disease of the kidney, and does not disappear after delivery.

7. Perhaps in puerperal convulsions, etc., produced by albuminuria, the immediate pathological cause of the nervous lesions is some unascertained but poisoned state of the blood. Was there a morbid quantity of urea in the blood? In several specimens of the blood of patients suffering under severe puerperal convulsions, furnished by Dr. S. to Dr. Christison and Dr. Douglas Maclagan, these gentlemen had been unable to detect any traces of urea. Was the poisoning material cascine in morbid quantity or quality? The dependence makes this connection worthy perhaps of some inquiry.

8. In cases of severe puerperal convulsions, etc., from albuminuria, the renal secretion is in general greatly diminished, and Dr. S. had found active diuretics apparently of great use along with or after venesection,

antimony, etc., especially where the case was offering to become prolonged.

9. Sometimes hemiplegia supervened during pregnancy without albuminuria, but this form did not seem to interfere materially or very dangerously either with the pregnancy or labor, the disease running its own usual course. In one case Dr. S. had seen the patient gradually but imperfectly recover the use of the palsied arm after delivery. In another no improvement occurred.

Garrod (*London Lancet*, 1848) says the presence of albumen in the urine, independent of blood or pus, is owing to some morbid condition of the kidneys.

Devilliers and Regnault (*Arch. Gén. de Méd.*, t. xv., xvi. et xviii., 1848) verified the coincidence of eclampsia and albuminuria. They report eight autopsies of cases of puerperal eclampsia, in four of which a disease of the tissue of the kidney was found. This condition thus early they called *albuminous nephritis.*

Dr. Cormack (*Med. Times*, London, 1850) also pointed out the coincidence of eclampsia and albuminuria.

Dr. G. O. Reese (*London Medical Gazette*, 1851) says, "The presence of albumen in the urine must be regarded as significant. Continued albuminuria unconnected with lesion of the kidney is rare."

Frerichs (*Die Brightsche Nierenkrankheit und deren Behandlung*, 1851) attributed puerperal convulsions to Bright's disease, and was the first of the Germans to devote especial attention to the nephritis of pregnancy.

On p. 215 of the above-named work he says, "True eclampsia occurs only in pregnant women suffering with Bright's disease, and it bears to the latter the same causal relation as convulsion and coma to Bright's disease in general; it is the result of the uræmie intoxication, with which also in its mode of manifestation it agrees."

This, Bright's disease, he ascribes to two causes: 1st, the alteration of the blood crasis caused by pregnancy; and, 2d, the mechanical interference with the venous circulation in the abdomen. By *crasis* Frerichs seems to have meant an impoverishment of the material blood consequent on the drain of nutritive matters for the nourishment of the fœtus.

Carl Braun, in January of the same year (1851), declared that, "As a rule, *eclampsia vera puerperalis* is found intimately connected with *diabetes albuminosus*" (*Ueber Eclampsie und Albuminurie in der Zeitsch. d. G. d. Wiener Aertzte*, 1851, Bd. i., S. 57). And in his *Lehrbuch der Geburtshülfe*, etc., Vienna, 1857, he devotes a chapter to the subject, which was shortly afterwards translated into English by J. Matthews Duncan, of

Edinburgh, and is well known to the profession as *Braun on the Uræmic Convulsions of Pregnancy, Parturition, and Child-Bed.*

In this volume he says, "The coincidence of eclampsia and albuminuria is an undisputed fact, which is verified by the very numerous observations of Lever, Simpson, Devilliers, Regnault, Dubois, Danyau, Cazeaux, Cormack, Blot, Helft, Frerichs, Litzmann, the author, and many others, as also by daily observations. This has opened a new path to the knowledge and treatment of this most dangerous disease, so that the eclamptic convulsions of women during pregnancy must be considered to be identical with the fits of adults in general that are produced by uræmia in the course of acute Bright's disease. This I publicly declared in January, 1851, and at the same time Frerichs published his most convincing classical treatise on the subject. It must now be considered an axiom in theory as well as in practice."

Braun proceeds to prove his proposition by a long argument, from which I abstract only such as is based upon *post-mortem* examinations or the presence of albumen in the urine. Of forty-five cases published by him, fifteen died, but *post-mortem* examinations were made only in twelve. In seven of these Bright's disease was proven to be present by microscopic examinations, made by Wedl and others. In all the remaining cases the kidneys were *hyperæmic;* but from accidental obstacles no microscopic examination was made. But, as he says, "this cannot be considered any proof of the absence of Bright's renal exudation." On the other hand, the hyperæmia would seem to afford presumptive evidence of the presence of Bright's disease. Gustav Braun further communicated to him six cases with fatal termination, in three of which Bright's disease was demonstrated beyond a doubt. He further alludes to one case published by Lumpe, two by Hecker, four by Devilliers and Regnault, three by Simpson, two each by Sabatier and Hohl, one by Blot, Cohen, Wieger, Litzmann, and Crede, in all of which were found Bright's disease of the kidney after death. Hasse never saw eclampsia puerperalis without Bright's disease. Braun concludes with the statement, "There are above thirty cases known to me offering positive proofs of the intimate connection between Bright's disease and eclampsia, and they are far too numerous to admit of the opinion that there is only an accidental connection between the two being still entertained. Their value as arguments is very much enhanced by the circumstance, that in negative observations the histology of the kidneys was examined microscopically only in very rare instances."

He further states that in an observation by Oppolzer and himself, they prognosticated the outbreak of eclampsia two days before its occurrence

from the presence of abundant albuminuria, and from the blood containing a large quantity of urea. Also "Devilliers, Regnault, the author (*i.e.*, Braun), Litzmann, and Wieger have published a complete series of observations, in which albuminuria and exudation clots (casts?) occur along with Bright's disease during pregnancy, lead to spontaneous premature labor under different uræmic phenomena, and then terminate in speedy recovery, having no further injurious consequences, and producing no eclampsia. The circumstance that eclampsia does not occur in every case of Bright's disease during pregnancy, as Litzmann has very correctly remarked, can be accounted for by this result following only when the blood has been very considerably impregnated with the excrementitial elements of the urine, which always implies a profound, or at least extensive disease of the renal tissue." Alluding to the large amount of albumen and the large number of exudation casts covered with abraded glandular epithelium, already in part undergoing fatty degeneration, or even passing into detritus, "no one can really believe," he says, "as Litzmann has strikingly pointed out, that a disease in the kidneys furnishing such products has just been developed within the last few hours."

As to the causes of Bright's disease, Braun includes, among the more remote, the peculiar changes in the blood of pregnant women, the pressure of the pregnant uterus, and the stoppage of the venous blood in the kidneys caused thereby. Of cases of eclampsia, eighty per cent. occur in first pregnancies, in which, on account of the greater resistance of the abdominal walls, a powerful counter-pressure on the kidneys is generally produced. In cases of repeated pregnancy, the pressure connected with a plural pregnancy, with deformed pelvis, hydramnos, the large size of the fœtus, and a high position of the womb are frequently met with where eclampsia occurs. He says, further, that the cases in which albuminuria occurs during the first half of pregnancy cannot be referred to pressure on the renal veins, but may, according to Litzmann, be traced back to catarrhal irritation of the urinary passages, or, in cases of really existing Bright's disease, to a complication existing before the pregnancy. Neither of these two observers appear to have met puerperal eclampsia as early as has been observed by more recent observers, during the second month. Finally, he admits that "congestion of the venous blood in the kidney is not the only cause of Bright's disease; for among cases of eclampsia there are twenty per cent. of premature births; and abortions, even in the fifth and sixth months of pregnancy, having fatal terminations, have been observed by Velpeau, Harris, Pätsch, and others. It is reserved for future inquiries to answer the question, whether or not eclampsia of the first half of pregnancy

appears always eontemporaneously with Bright's disease." The fact that after the womb is evacuated, and at the same time the impediment to the free eirculation of venous blood is removed, the albuminuria disappears with great rapidity is pointed out as showing the intimate connection between the two, that is, the pressure and Bright's disease.

The name of Litzmann is conspieuous in the history of the relations of puerperal eclampsia to Bright's disease. He early entered the lists in support of the dependence of the former upon the latter. In his first paper (*Deutsche Klinik*, 1852, No. 19, *et seq.*) he reports, first, six eases of parturient women in whieh there were symptoms of Bright's disease, as shown by albuminuria, diminished seeretion of urine, uræmia, in all of which, exeept one, eelampsia oeeurred. The latter was that of a woman in whom, in the first pregnaney, there were the symptoms of Bright's disease *with* eonvulsions. In her second pregnancy there were the same symptoms, ineluding albuminuria, uræmie intoxication, and amaurosis, but without eonvulsions; the albuminuria disappearing after delivery. In a second series of six eases there were the same symptoms of Bright's disease without eelampsia. One of these eases died, revealing evident Bright's disease of both kidneys.

Litzmann agrees with Rayer and Lever as to the eause of the albuminuria and Bright's disease of pregnaney. He attributes them to meehanieal pressure and to stagnation of eireulation, especially in the kidneys. He observed the development of albuminuria most eommonly in primiparæ (in which the abdominal muscles do not yield so easily to pressure as in the multiparæ), or in multiparæ with narrow pelves, or in twin pregnancies, or a very large child, or hydramnos. In three eases of albuminuria the women were affected with a ehronie eatarrh of the lungs; the impaired pulmonary eireulation might have given rise to a venous hyperæmia of the kidneys. The frequent instantaneous disappearanee of the albuminuria with the emptying of the uterus, which was pointed out by Rayer, is also in favor of the theory of meehanical pressure.

Litzmann believes with Lever, Devilliers, and Regnault that the usual existing eauses of Bright's disease in the non-pregnant, such as eold and exeess in aleoholie drinks, eannot operate in produeing the Bright's disease of pregnaney. He also does not believe that mental distress and sorrow ean favor the development of the disease.

The lesion of the kidneys found by Litzmann eorresponded mostly with those of an early stage of Bright's disease. In his experienee, Bright's disease never develops itself in the pregnant woman before the eighth month; but he alludes to two eases observed by Devilliers and Regnault, where it

began in the sixth month, and produced eclampsia. This, however, he considers the earliest possible period, as before the sixth month the uterus is hardly large enough to exercise any pressure on the renal veins, and where albuminuria exists earlier, he attributes it to a catarrhal irritation of the ureters and bladder with purulent secretion, which he was the first to describe, or to chronic Bright's disease existing before the pregnancy.

For the diagnosis of this affection, he considers the only reliable signs to be albuminuria and tube-casts. Œdema is often wanting. He also accepts Frerichs's view, now very generally given up, that it is not the retention of urea in the blood which causes the eclampsia, but carbonate of ammonium, derived from a decomposition of the urea. Litzmann considers that his observations confirm this theory.*

In a second paper, published also in the *Deutsche Klinik*, 1855, Nos. 29 and 30, Litzmann's object is to support the views advocated by him in 1852 against the attacks of Scanzoni and others. More cases are reported, including those of Simpson, Hecker, Lumpe, and Braun, already given in the abstract of Braun's views. Also another by Litzmann himself, in which the first pregnancy was attended by eclampsia; the second and third were normal; fourth, eclampsia; fifth, no eclampsia, but amaurosis; sixth, eclampsia at sixth month; seventh, eclampsia; eighth, abortion at second month; ninth, pregnancy terminated safely, although Bright's disease was evidently present, the urine being loaded with albumen and containing tube-casts. All the pregnancies were accompanied by œdema and temporary suppression of urine. Perspiration, however, was increased at these times. The inference of Litzmann was that a Bright's disease started during pregnancy may often continue as a chronic disease after delivery.

E. Robins (*London Lancet*, 1852) says, "Albumen appears in the urine during gestation where sufficiently advanced to occasion a habitual congestion of the kidneys."

In 1856, Imbert-Goubeyre published his thesis *De l'Albuminurie Puerpérale et de ses Rapports avec l'Eclampsia* (Mémoires de l'Académie Impériale de Médecine, Paris†), crowned by the French Academy. In this he says, "It is at present no longer possible to deny the connection

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* It may be remarked here that this theory of Frerichs's has been again brought forward by Spiegelberg (*London Lancet*, December, 1870), founded upon the result of a series of experiments conducted by himself and Heidenhain.

† Although this is the date of publication of Imbert-Goubeyre's paper, it was probably written between 1850 and 1855, as it is quoted freely as a "Manuscript Memoir" by Cazeaux, in the fifth edition of his Midwifery, the preface of which is dated November, 1855.

of eclampsia with albuminuria, or Bright's disease. Taking all facts together, true eclampsia is nothing but Bright's puerperal disease in which convulsions prevail: it is Bright's disease accompanying pregnancy, and it predominates then with a particular form of cerebral accident, which is eclampsia. This is proved by its symptomatology, its progress, its termination, its prognosis, and its anatomical lesions."

Cazeaux early examined the kidneys from fatal cases of puerperal eclampsia with Rayer, but the earliest publication of his views which I find is in the second American from the fifth French edition of his Midwifery. The latter was issued in 1855.

He says at the outset (p. 289 of the American edition), that as albuminuria is generally the symptom of an organic disease of the kidneys which almost always proves fatal, it becomes important to determine whether its presence in pregnancy is due to the same cause, or whether it is merely one of the numerous modifications produced in the economy by gestation; for, in the first place, it is a serious condition, and in the second, a temporary functional disorder which will most probably disappear with the cause.

In answer, he says, "Observation shows that in almost all the cases in which women die of the convulsions which too frequently complicate albuminuria, the kidneys present the anatomical characteristics of albuminous nephritis, the more or less advanced degrees of alteration appearing to correspond with the duration of the disease and the amount of albumen discharged. Many times have I had occasion to observe this fact, and fearing lest I should interpret the alterations erroneously, have almost uniformly presented the kidneys to the examination of M. Rayer, who generally recognized in them the second, sometimes the third, and only once the fourth degree of alteration."

"The learned physician of La Charité considers the more frequent occurrence of the anatomo-pathological characters of the second degree of the disease to be due solely to the recency of the latter, and by no means to a difference of nature. It is no less the consequence of a renal hyperæmia, which he supposes may be caused in many cases by compression of the emulgent veins by the enlarged uterus, and the consequent obstruction to the return of venous blood. That, in simple cases, it generally disappears promptly after delivery is probably due to the consequent cessation of the congestion of the kidney, which was maintained by the pregnancy."

Cazeaux says, further, that many of the objections to this view may be met by the fact first observed by M. Pidoux, and too often ignored, "That the secretion of urine is not confined to the kidney, since it takes place

previous to the formation of the latter. The proeess of assimilation, which is so active in the fœtus, ean only be understood by supposing a contemporaneous proeess of deeomposition. The blood which flows to the organ is already eharged with the elements of urine, which are to be separated from it in the passage. The function begins in all parts of the eeonomy by this admixture of heterogeneous elements with the blood, and is eompleted in the kidney by their elimination from the eireulating fluid, which is returned in a purified eondition. . . . To study the latter organ exclusively, when we wish to obtain a physiologieal idea of the function, is to negleet an important element; so, also, always to expeet to find the cause of the disorders of the urinary seeretion in alterations of the kidney is to overlook a multitude of other eauses whieh have a eorresponding influenee. The elements of the blood eonveyed by the renal artery exist in health in a fixed proportion, and eertain of them are destined to be eliminated by the kidneys. Now, it is easy to understand that if an alteration in the strueture of these organs is eapable of modifying both the quantity and quality of the matters eliminated, an alteration of the fluid, such, for example, as the diminution or inerease of its solid or fluid parts, may have the same effeet. Clinieal observation and *post-mortem* examination give eonstant support to this idea; for, though we sometimes find a material lesion of the kidney to which we attribute the albuminuria, we are very frequently obliged to reeognize it also in eertain general diseases, as searlatina, typhoid fever, eholera, etc., in whieh the renal affection is often wanting. In Bright's disease itself, or at least in eertain eases in whieh the presenee of albumen in the urine, and all the other symptoms of granular nephritis, had been deteeted during life, the *autopsy*, though eondueted with the greatest eare, revealed no alteration of the kidneys (Forget, of Strasbourg). Finally, Kennedy found the kidneys to be perfeetly healthy in several eases of dropsy with albuminuria following scarlatina. Must we eonelude from these that Bright's disease did not exist in those eases? Certainly not; the disease was present, but the pathologieal alteration was eonfined to the fluids."

"This," he eontinues, "may be the ease in pregnaney." "The eomposition of the blood is elearly altered. It eontains less albumen, fewer eorpuscles, more fibrin, and, aeeording to some, also more urea. Its influenee on the kidneys eannot be denied, varying with the duration and degree of alteration. If the latter be slight, the effect will be almost nothing; if more prolonged or intense, it will eause greater derangement of function, indieating its existenee by albuminuria and anasarca. All this may take plaee *without any appreciable lesion of the kidneys.*"

He considers the mistake of M. Blot and others who insist so strongly upon the cases of puerperal albuminuria without renal alteration, with the object of proving that the presence of albumen is but very rarely the consequence of Bright's disease, consists in their confining Bright's disease to the renal alteration exclusively. We may have *variolæ sine variolis*, —typhoid fever without the intestinal eruption; why not Bright's disease without renal alteration?

Such is the argument of Cazeaux with regard to the relation of albuminuria and pregnancy. As to the relation of albuminuria to eclampsia, he says first (p. 716, *op. cit.*), "Since the presence of albumen is discovered almost constantly in cases of eclampsia, the severest mind can hardly avoid establishing a more or less intimate relation of causality between the two facts; . . . and since albuminuria is present in the immense majority of eclamptic women, it, or rather the disease of which it is a symptom, may be rightfully regarded as the predisposing cause of eclamptic convulsions."

The fact that the amount of albumen is markedly increased during the eclamptic fit and generally diminishes after it, which is used by some to prove that albuminuria is the result of eclampsia rather than the cause of it, he disposes of practically by denying the possibility of eclampsia occurring without being preceded by albuminuria. He says, "I can understand why there might be hesitation in regard to this point, if a single case could be cited in which it had been proved that the urine was entirely free from albumen for several weeks before the appearance of the accident. This, I believe, has never been done, but often, on the other hand, albuminuria has been known to be present for some time before the convulsions occurred." It is easy to understand, also, how a renal congestion may take place in the kidneys in common with other internal organs during labor, and the increased secretion of albumen should result from such congestion. He admits also that the condition of the blood which results from a long-continued albuminuria, by giving rise to a peculiar excitement of the cerebro-spinal centre, may itself become the direct cause of the convulsion, or, at least, which is more frequently the case, render it more susceptible to the excitements which reach it either from without or from previously irritated internal organs.

In speaking of the pathological anatomy of the disease, he says, "What we have stated in regard to the almost uniform coincidence of albuminuria with eclampsia, and to its common connection with lesions of the kidneys, sufficiently indicates that the anatomical lesions are hereafter to be sought in those organs. For our own part we have never failed to do so for the

past ten years, nor do we hesitate at the present time to consider albuminous nephritis as one of the most common lesions after puerperal convulsions. As already stated, the kidneys have almost universally presented the anatomical characters of nephritis, the more or less advanced degrees of which appeared to coincide with the chronicity and abundance of the albuminuria.

"Other observers, amongst whom I may mention MM. Blot and Depaul, state that usually they have met with no disease of the kidney, and, regarding the above-mentioned facts as altogether exceptional, insist that in the majority of cases Bright's disease has no connection with eclampsia.

"In the first place, I would call attention to the fact that I do not regard Bright's disease as residing in the lesion of the kidneys exclusively (page 294), and although the kidneys should present nothing abnormal, the alteration of the urine is sufficient to prove its existence. I might, therefore, strictly pay no regard to the facts mentioned by my opponents; but let us examine whether, independently of the opinion which I support, the observations of MM. Blot and Depaul are of much value. They have found nothing, say they; but perhaps their not having done so is their own fault in not having examined sufficiently, and I have to acknowledge that hitherto I had committed the same error. Works recently published in Germany show, in fact, that the naked eye is entirely incompetent to detect anatomically the commencement of albuminous nephritis, and that the first degrees of renal alteration can be discovered only by the microscope."

W. R. Basham, M.D., in his book on Dropsy (1858), says that the albuminuria in pregnancy is due to the obstruction of the circulation in the kidneys caused by direct pressure of the gravid uterus. The albuminuria may pass away after delivery, but sometimes, if the pressure is prolonged and excessive, it leads to true kidney-disease, in which case there are also found tube-casts.

Dr. Krassing in *Spitals Zeitung*, 1859, reports nineteen cases of eclampsia which occurred in the clinic of Braun, in Vienna, in 1857 and 1858. Nine died. In all the nineteen cases save one there was reason for believing that Bright's disease was present. In one case with sixty paroxysms, no albumen was found in the urine. In ten cases there was œdema. In one case where eighty-one attacks were observed there was no œdema. Of the cases examined after death, there was found hyperæmia of the kidney with inflammatory exudation in three cases, fatty degeneration in four cases, atrophy in one case, and no morbid change in one case.

Dr. Perrie (*Dubl. Qu. of Med. Sci.*, vol. xxix , 1860) says albuminuria is most probably caused by renal congestion, the effect of direct pressure.

Dr. L. X. Bourgeois, in a memoir (*De l'Influence des Maladies de la Femme pendent la Grossesse sur la Constitution et la Santé de l'Enfant*) read before the Imperial Academy of Medicine, in 1861, says puerperal eclampsia is due to Bright's disease.

Dr. Bosse (*Spitals Zeitung*, 1862) relates fourteen cases of eclampsia which occurred within thirty-one months among four thousand five hundred women at the Lying-In Hospital at Gratz. Of these fourteen cases, thirteen were primiparæ; eight recovered and six died. In *all* these last there was intense Bright's disease, and in five cases œdema of the lungs.

Bedford (*Principles and Practice of Obstetrics*, 1861, p. 506) says, "Albuminuria is due to the following causes: 1st, a change in the composition of the blood; 2d, a change in the kidneys, either structural or dynamic; 3d, pressure of renal veins."

The late Prof. George T. Elliot (*Obstetric Clinic*, 1868) reports a large number of cases of puerperal albuminuria and eclampsia, in many of the fatal cases of which *post-mortem* examinations revealed lesions of the kidneys; and although he does not state explicitly his views with regard to their necessary relationship, it is evident he inclines to this view. His colleague, Prof. Barker, however, says, on p. 104 of *The Puerperal Diseases*, "Another would only make two classes, the uræmic and hysterical. My friend and colleague, Prof. Elliot, followed this last division, calling those convulsions which were general in their character, and attended with loss of consciousness, eclampsia; and he believes that these were always associated with renal lesions."

Bailey (*Dictionnaire de Méd. et de Chirurgie*, "Eclampsia," 1870) says, "The only organic lesion in eclampsia is that of the kidneys; it consists of a congestion, of desquamation of the epithelium of the tubules, fibrinous exudation into the tubules and into the parenchyma of the kidneys, fatty degeneration of the elements, and, in rare cases, glandular atrophy, which forms the highest anatomical expression of a chronic form of Bright's disease."

M. Moss (*Prog. Méd.*, 1873) found the kidneys, in a case of eclampsia, to present the characters of *parenchymatous nephritis*.

Leishmann (*System of Midwifery*, 1873, p. 475) says, "With rare exceptions, then, arising from these or similar causes (epilepsy, diseased conditions of nerve-centres, and hysteria), puerperal eclampsia may be looked upon as essentially connected with uræmic poisoning, which, again,

is associated with or dependent upon an albuminous condition of the urine." And as to the cause of the renal lesions, he believes (p. 254) that it is due to the pressure of the gravid uterus in many instances, while he is inclined to think that this explanation has been too readily accepted as the solution of the case, especially in cases where albuminuria has appeared so early that pressure is impossible. Without pretending to account for the condition in these cases, he quotes Dr. Tyler Smith thus: "In such cases," says Dr. Smith, "the disease appears to me to depend upon reflex irritation of the kidneys by the gravid uterus, similar to the irritation of the salivary glands, the mammæ, thyroid, etc., and not upon mere pressure alone."

Dr. Depaul (*Archives de Tocologie*, 1874) reports several cases of eclampsia in the sixth month of gestation, with symptoms of uræmic poisoning and albuminuria, and total absence of œdema.

Dr. Johnson (*Lectures on Bright's Disease*, 1874, p. 118) admits that puerperal eclampsia may occur without albuminuria, and, on the other hand, albuminuria in pregnant women may be unassociated with convulsions. He limits his discussion, however, only to puerperal albuminuria, and, referring to illustrative cases, he arranges the latter into four classes:

1. Cases of women suffering from chronic Bright's disease, who passed through all the stages of parturition many times *without eclampsia;* free secretion of urine continuing, and preventing it by maintaining elimination.

2. Cases (mostly primiparæ) where the *pressure of the gravid* uterus on the vena cava produced a passive renal congestion, albuminuria, and uræmic *convulsions.* He argues that in multiparæ the abdominal walls are more yielding, and pressure is thus avoided.

3. Cases of *acute desquamatic nephritis,* originating during pregnancy from exposure to cold and wet, drinking, or from zymotic blood poison, or which may be due to some blood-change, to the evolution of the uterus and the growth of the fœtus. The renal symptoms may pass away after delivery; they may or may not return with the next pregnancy. In cases where the albuminuria is persistent, ultimately uræmic symptoms occur, and after death the kidneys are found either *contracted and granular* or *large and pale.*

4. Cases in which albuminuria and convulsions appeared for the first time *after delivery.* The urine is in such cases indicative also of an *acute desquamatic nephritis;* these cases are pathologically allied to, and sometimes associated with, a form of septicæmic puerperal fever. The

cause of the renal symptoms here is "the blood-contamination consequent on the absorption of morbid materials from the interior of the uterus after parturition." The renal symptoms may pass away, or the disease may become chronic and terminate in a *large white kidney.*

Dr. M. Peter, in a series of lectures on puerperal eclampsia, their causes, nature, and treatment, published in the *Archives de Tocologie* for 1875, first described, not very precisely, the *post-mortem* appearances of the kidneys of two primiparæ dying in puerperal convulsions. The organs were somewhat enlarged and very anæmic; the cortical substance presented the appearance of cooked veal, and the pyramids were hyperæmic. In the second case, in addition to the above, he says the kidney corresponded anatomically with the third form of Bright's disease, described by Rayer. The injection at certain points was so great that the veins were distended with blood-clots. There was also albuminuria, and the microscope revealed numerous blood-globules, epithelium desquamated from the uriniferous tubules, and granular casts. "In brief, then," he says, "our eclamptic had had Bright's disease of puerperal origin."

The next lecture he begins by saying, "All pregnant women attacked with eclampsia are albuminuric. Such is the proposition formulated by Cazeaux, confirmed by Frerichs, by a professor in Vienna, Braun, and by all those who have studied eclampsia in the pregnant woman. . . . To this proposition (and for the great glory of a certain theory) some have opposed this argument, that there are pregnant women attacked with eclampsia who are not albuminuric. And they have cited in favor of this opinion as many as *six* cases,—*six cases* against the hundreds of cases of albuminuric eclampsia!"

Doubtless, he admits, there are exceptions, but these cannot change the rule, and it must be remembered that a large number of pregnant women having albuminuria may lose it temporarily. This is beautifully shown by Bailey in his article on "Eclampsia," in *Dictionnaire de Médecine et de Chirurgie Pratique.* He has observed that not rarely albuminuria in pregnant women disappears for several hours, but subsequently reappears again; and the cause of this may be attributed to a congestion which may suddenly vary in degree or disappear. The six cases in question he thinks cannot be explained otherwise than that the examination was made during the short period when the urine ceased to be albuminous. "I insist," he says, "upon my original proposition,—'all pregnant women attacked with eclampsia are albuminuric.'"*

* Dr. Peter prefers, not inaccurately, to speak of the passage of albumen into the urine as a "transudation of serum," rather than of albumen from the blood.

He says further that an analysis of the urine shows, as we would expect, a greatly increased elimination of urea during the pregnant state. The analyses of Quinquaed have shown that the pregnant woman, instead of secreting 22 to 24 grammes of urea in the twenty-four hours, eliminates 30 to 38 grammes,—nearly twice as much. This uropoiesis for two of course necessitates the movement of an increased amount of blood through the kidney, and produces a functional hyperæmia and a consequently increased intra-vascular pressure, which facilitates what he calls not merely an albuminuria, but a *serumuria*. Further, by the microscope we are enabled to judge of the exact condition of the kidney. The presence of renal epithelium of granular and hyaline casts shows that in the situation where the desquamation has taken place the kidney has absolutely lost the power of secretion: it has become a mere passive organ, through which the serum filters as it would through filter-paper. Hence the integrity of the kidney may be said to be inversely proportional to the quantity of albumen. With such defective secreting power, the urea must accumulate in the blood. But there is not only an accumulation of the urea, but also of all the constituents of the urine. Analyses of the blood of eclamptic patients have shown three times the quantity of urea found in normal blood. But the extractives have also been found increased threefold; so with the creatinin; and we should not therefore speak of an uræmia so much as of an urinæmia. Such a condition Dr. Peter likes to speak of as an *autotyphization* of the blood, or of a *typhization urinemique*.

Dr. Peter proceeds further to fortify his views as to the pathology of puerperal convulsions by the observations, familiar to most of us, of Dr. A. Mahomed, made in the Fever Hospital of London, and published in the *Transactions of the Royal Medico-Chirurgical Society of London* for 1874. These were to show that there was a like increased arterial tension in the vascular system of the pregnant woman, and in what he calls the pre-albuminuric stage of Bright's disease, during which the crystallizable principles of the blood transude into the uriniferous tubules before the albumen. Such transudation Dr. Mahomed proved by the guaiacum test,—the tincture of guaiacum striking with urine under these circumstances the characteristic delicate blue color of the coloring-matters of the blood,—while there is still no albuminuria. The increased tension was shown by sphygmographic tracings. The same condition is proven by the miliary aneurisms observed by ophthalmologists and the hemorrhages into the eyeball which have occurred under these circumstances. Finally, he considers them further sustained by the peculiar views of the physiologist Küss on the secretion

of urine, who believes that in health the serum of the blood, with its contained albumen and salts, is transuded into the Malpighian capsules, and that the albumen is reabsorbed by the epithelium of the convoluted tubules.

Those who claim that the phenomena of puerperal eclampsia are due to deranged circulation, the result of pressure upon the renal veins and inferior cava, are asked to account for the convulsions which present themselves in the early months of pregnancy, before the uterus has risen above the cavity of the pelvis; while, unfortunately for this theory, we see women who have enormous cysts of the ovary who have neither eclampsia nor albuminuria; others with enormous fibromata of the uterus without any trouble with the renal secretion. The pressure, according to Dr. Peter, is within the blood-vessels of the kidney, and not without. Dr. Peter, moreover, does not forget to say that not all albuminurie women are necessarily attacked with eclampsia.

Dr. Bourneville, in the *Archives de Tocologie*, 1875, p. 194, reports four cases of puerperal eclampsia, in all of which albuminuria was present. Two terminated fatally, but a detailed record of one case only is furnished. She was a primipara; the urine was loaded with albumen, and contained granular tube-casts and red blood-disks. Convulsions set in during labor. The *autopsy* revealed incipient putrefaction on the anterior parts of the kidneys. In other places the tissue of the organ was pale, except the cortical substance, which was yellowish in hue. There was no notable injection, except a little black blood in the larger vessels. The description lacks preciseness, but associated with the albuminuria Bright's disease may be inferred.

Dr. Eugène Marchal (*Archives de Tocologie*, 1875, p. 690) reports three cases of puerperal eclampsia which terminated in death: 1*st case.*—Multipara: urine dark-colored, highly albuminous, and the microscope revealed presence of blood. *Autopsy.*—Kidneys hyperæmie; hypertrophy of the epithelium of the tubules, with beginning fatty infiltration; no alteration in the connective tissue, nor any deformation in the tubules (first stage of Bright's disease).

2*d case.*—Primipara: unmarried; albuminuria. *Autopsy.*—Kidneys highly congested, chiefly in the cortical substance; the epithelium of the tubules hypertrophied and fattily infiltrated (latter stage of Bright's disease).

3*d case.*—Primipara: unmarried; delivery normal of healthy child; *no albumen in urine before delivery*, but as soon as eclampsia set in *albumen* was found in the urine copiously. Twenty-four hours later the patient died (having had twenty-two attacks). *Autopsy.*—The kidneys

strongly hyperæmic; the epithelium of the tubules hypertrophied, but no alteration in the connective tissue, and no change in the shape of the tubules. Liver fatty.

Bartels, in his article on the "Acute Parenchymatous Nephritis of Pregnancy," in Ziemssen's *Cyclopædia of Medicine*, 1877, says, "Apart from hemorrhagic extravasations,—which are wanting, too, in a great many cases of scarlatinal nephritis,—the pathological changes in the renal disease of pregnancy are identical in every respect with those of the other forms of acute nephritis." Also, "When we study carefully the descriptions of the microscopic appearances of the kidneys removed from the bodies of lying-in women who have died of eclampsia, a number of which were published by Litzmann in the *Deutsche Klinik* for 1855, we find that they correspond exactly with the appearances which, as we saw in the preceding chapter, are presented by the acute renal inflammations due to other causes; for instance, by scarlatinal nephritis that has run a protracted course. The kidneys are larger and heavier than normal, from thickening of the cortical substance. The cortex is anæmic, of a pale yellowish color, and of a swashy consistency. What resemblance is there here to the very vascular, dark-colored, and usually firm and compact kidneys that we find where venous stasis has existed?* It is unnecessary to add anything concerning the changes in the other organs of the body that are found at the *autopsy*. I will only mention that dropsical effusions are seldom altogether absent. Thus, Braun found œdema in thirty-nine out of forty-four cases of eclampsia."

On p. 313 of the same article, Bartels also says, "That acute uræmia occurs more frequently in the nephritis of pregnancy than in the other forms of this renal disease will be admitted even by those who, like myself, are not inclined to regard every case of eclampsia or mania occurring during parturition as of uræmic origin, even although the urine, after the outbreak of the symptoms, contains some albumen."

Bartels contends strongly against the theory of renal pressure adduced by Lever and Rosenstein as causes of the nephritis. He says, "It must at once strike the student that not a single iota of evidence founded upon fact can be adduced to establish this altogether gratuitous assumption of a compression of the renal veins by the pregnant uterus. The clinical symptoms are by no means the same as those of obstructive engorgement, and the anatomical appearances not only do not resemble those of cyanotic induration of the kidneys, but correspond in every particular with those of

* Interstitial nephritis is here alluded to.

parenchymatous nephritis." He further says that the position of the renal veins protects them, under all circumstances, from any direct pressure on the part of the pregnant uterus, and any one who has had the opportunity of opening the body of a woman far advanced in pregnancy, and has observed the relative position of the abdominal organs, could not fail to perceive how inadmissible is the theory that a mechanical pressure is exerted upon the renal veins by a distended uterus. He then proceeds to demonstrate the anatomical impossibility of such pressure occurring, for which reference must be made to his article.

With regard to the actual cause of the parenchymatous nephritis, except to say that there must be some special cause in the pregnancy itself, he does not bring forward any, but says that, "For the present there is nothing left for us but to record the fact that parenchymatous inflammations of the kidneys and liver (as originally shown by Virchow with regard to the latter organ) may be developed during pregnancy, and to confess that we do not know what causes them."

Dr. W. Howship Dickinson (*On the Pathology and Treatment of Albuminuria*, 2d ed., London, 1877, p. 158) says, "Another condition—acting probably mechanically, and producing, as does heart-disease, venous congestion of the kidney, and the train of organic changes and constitutional results which spring therefrom—is pregnancy.

"The disproportionate results, as compared with other forms of renal disease, particularly in respect to convulsions, which have been stated to occur in one-fourth the cases of albuminuria of pregnancy, may be perhaps due to the disturbing influence of labor, the exaltation of nervous susceptibility which would seem to accompany the act, or the effect which it has, whether by exhaustion or febrile action, of destroying the equilibrium of the nervous system. Thus, under the sudden influence of parturition, uræmic convulsions may result from an amount of uræmia which might otherwise be inadequate to produce them."

Julius Althaus, in his recent work on *Diseases of the Nervous System* (G. P. Putnam's Sons, New York, 1878), has an excellent article on puerperal eclampsia, in which he says, referring to the view of "Scanzoni and some other high obstetric authorities," who attribute the albuminuria of pregnancy to venous congestion, produced by an attack of eclampsia as the primary disease, "This view is most certainly not borne out by the facts of the case. In the first place, we have to notice the physiological fact that removal of both kidneys in animals gives rise to uræmia and convulsions. Secondly, it is contrary to fact that convulsive attacks tend to produce albuminuria, because even after a long series of epileptic attacks

no albumen is found in the urine. Thirdly, the disease is not mere albuminuria, or passive transudation of albumen from congestion, but real nephritis, as shown by the microscopic appearances of the urine during life, and of the kidneys after death. Fourthly, this nephritis is almost invariably prior to the convulsions; for although in some well-observed cases albumen has been absent from the urine before a fit, and present after one, it seems that œdema has always preceded the fit. Now, it is well known that albumen in the urine is not always the first symptom of nephritis: for in certain cases of scarlatina, dropsy has been observed before the albuminuria."

As to the cause of the nephritis before and after parturition, he says it is obscure, but brands the pressure theory as erroneous, for reasons practically the same as those already given; also, that in the absence of any better reason we may consider both the nephritis and eclampsia due to the circulation of some irritant material in the blood. He does not even admit the occasional operation of one of the usual causes of nephritis, as cold, excess in drinking, etc., for the reason that the disease generally vanishes after the lying-in period.

I have not had access to the work of Prof. Hervieux, physician to the Maternité Hospital, Paris, *On Puerperal Diseases*, but Prof. Barker in his own book on this subject says Prof. H. "seems to regard puerperal albuminuria as mainly caused by what he calls 'puerperal poison,' and as analogous to the albuminuria which occurs from the scarlatinal poison."

From the standpoint of pathological anatomy, Rindfleisch, in his *Pathological Histology* (1873), treating of acute parenchymatous nephritis, mentions "puerperal processes" as one of its causes.

Cornil and Ranvier (*Pathological Histology*, 1876, p. 1035) say, "Nephritis with albuminuria being more or less persistent, but generally curable, is observed in the course of pregnancy, or during or after delivery, and may or may not be accompanied by *eclampsia;* the lesion of the kidneys consists of a granular fatty degeneration of the epithelium comparable to that observed in scarlatina."

In a letter to the writer, dated May 5th, 1878, Prof. T. Gaillard Thomas, of New York, says, "During a practice of twenty-seven years in a large city, and connected with large hospitals during the whole period, I have of course seen a great many cases of puerperal convulsions. On two occasions only has albumen been absent from the urine in all these cases. In one it appeared two or three days after the convulsive seizure, in the other it never appeared.

"My belief is that puerperal convulsions are very generally due to poison

of the blood by elements of the urine, but that in exceptional cases any central or puerperal irritation which would create similar seizures in a child will produce them in the pregnant or parturient woman."

Dr. Ellwood Wilson, of Philadelphia, under date April 1st, 1878, writes, "I have seen no case of puerperal convulsions without albumen in the urine, although on some occasions when I had inquired of the attending physician whether there was albumen in the urine, and have been informed there was none, upon passing the catheter and testing the urine it was found to be heavily loaded with albumen."

Prof. Theophilus Parvin, of Indianapolis, writes April 18th, 1878, "In every case in which I examined the urine, there was found albumen."

Dr. Edw. L. Duer, of this city, has kept record of but five cases out of six which came under his observation. "In all of these cases there was present more or less albumen."

II. *Authors whose observations tend to controvert the view that puerperal eclampsia is due to Bright's disease.*

The theory that puerperal convulsions are reflex actions excited by cerebro-spinal or medullary irritation, of uterine origin, and transmitted through the ganglionic cells in which the reflex nerves terminate, is traced back by Dr. Thomas Moore Madden, in a paper read before the Obstetrical Society of Dublin, May 9th, 1874, to Laurence Joubert (*De Convulsionis Essentiâ et Causis*, op. An., p. 219, Ed. Antwerp, 1500), who was Professor of Medicine in the University of Montpellier in the middle of the sixteenth century. He asserted that the cause of convulsions is irritation, and that only by the removal of the cause of this irritation can the paroxysm be arrested.

The older English and American obstetricians generally claimed that puerperal convulsions were due to a determination of blood to the head, to be treated by blood-letting. This was the view of Scott (*Lectures on Midwifery*, 1775), Smellie (*A Collection of Cases and Observations in Midwifery*, 1779), Denman (*Introduction to Midwifery*, 1781), Foster (*Principles of Midwifery*, 1781), Bland (*Human and Comparative Parturition*, 1794), the Hamiltons (*Treatise on Midwifery, Hamilton's Lectures on Midwifery in the University of Edinburgh*, 1815–16. Reported by Dr. McKeever, MSS.), William Hunter Ryan (*A Manual of Midwifery*, 1831), Blundell (*Principles and Practice of Obstetric Medicine*, 1831), Davis (*Principles and Practice of Obstetric Medicine*, 1836), Burns (*Principles of Midwifery*, 1843), Maunsell (*Dublin Practice of Midwifery*, 6th Edition. Edited by M. Madden, M.D., 1871, p. 194).

Dr. Inglis (*Facts and Cases in Obstetric Medicine*, 1836) and others claimed that the circumstance that eclampsia commonly begins at night is a proof that the disease is connected with congestion of the brain.*

W. P. Dewees, M.D., in his *Essays Connected with Midwifery*, Philadelphia, 1823, and *System of Midwifery*, 1833, divides, as do several other older authors, puerperal convulsions into three different kinds: the epileptic, apoplectic, and hysterical species, according to the character which the convulsions bear. In his experience the "robust and plethoric women, and chiefly those of the lower class," are more liable to this disease, whilst "delicate and relaxed" women are seized generally with the hysterical species of puerperal convulsions, which occur less frequently and are less dangerous than the other species.

Dewees did not attempt to explain the cause of eclampsia, nor did he trace it to any causative lesion or to any changes in the blood.

He does not admit that pressure of the gravid uterus upon the descending blood-vessels, "causing a regurgitation of blood to the head, could produce convulsions, as this pressure is uniform in every pregnancy, and convulsions are comparatively of rare occurrence." He thinks, also, that pregnant women may be seized with convulsions from other causes than gestation.

Dr. Tyler Smith in his lecture on the cause of puerperal convulsions (*Parturition, and the Principles and Practice of Obstetrics*, 1849, p. 282), the substance of which was published in the London *Lancet* in 1844–45, before giving his own views presents those of cotemporary writers in England.

Ramsbotham (*Principles of Obstetric Medicine and Surgery*, 1842) says, "The most usual *proximate* cause of puerperal convulsion is probably *pressure on the brain*, this pressure being sometimes produced by the rupture of a vessel causing a sudden effusion of blood; sometimes by serous exudation into the ventricles or between the membranes; sometimes, and by far the most frequently, by *simple congestion of the cerebral vessels themselves*. But the disease has often proved fatal without any organic lesions being evident on dissection, and without even the vessels being observed to be preternaturally full. Into the *remote* causes it is not my wish to enter at any length, *because the subject is at best unsatisfactory, and little understood*. They have been ascribed to articles of food remaining undigested on the stomach, or irritation existing in some other

* For the facts in this paragraph I am indebted to the paper of Dr. Madden referred to, which is published in vol. ii. of *The Obstetrical Journal of Great Britain and Ireland*, April, 1874, to March, 1875, p. 236.

part of the alimentary tube; to general irritability of constitution; to a delicate and luxurious mode of living; to the depressing passions; to an overloaded state of the system; to over-distention of the uterus; to distention of the bladder; and to the death of the child. But the affection, in my opinion, originates most frequently in some deranged state of the uterus itself, probably in its nervous system, and consists in some irritation propagated from that organ to the brain."

Dr. Rigby (*System of Midwifery*, 1849). "The exciting cause of *eclampsia parturientium* is the irritation arising from the presence of the child in the uterus, or passages, or from a state of irritation thus produced continuing to exist after labor. The predisposing causes are general plethora; *the pressure of the gravid uterus upon the abdominal aorta;* the contraction of that organ during labor, by which a large quantity of the blood circulating in its spongy parietes is driven to the rest of the system, constipation, deranged bowels, retention of urine, previous injuries of the head, or cerebral disease, and much mental excitement.

"Also, in persons of hereditary predisposition, spare habits, irritable temperament, high mental refinement, and in whom the excitability of the nervous and subsequently the sanguiniferous system is called forth by causes apparently trivial."

Burns (*Principles of Midwifery*, 1837). "Convulsions of the kind I am considering evidently are connected with gestation or parturition; they occur at no other time, and are more frequent in a first labor. . . . They arise particularly from uterine irritation, but also seem frequently to be connected with a neglected state of the bowels. . . .

"A sympathetic irritation is almost invariably accompanied by an affection of the vascular system, productive of *great determination to the head*, either directly or indirectly, through the medium of the spinal nerves, which aggravates the evil, and becomes, indeed, the chief source of danger. I am inclined to think that, in a majority of instances, *the spinal cord is affected* by the state of the uterine nerves, and immediately afterward the head suffers. . . .

"A strong predisposition is given to this condition of the nervous system by a bad state of the bowels; and labor seems to bring the matter to a serious crisis. . . . On inspection after death, we sometimes find turgescence of the vessels of the brain or slight effusion of serum, but very often no mark of disease is to be discovered anywhere."

Dr. Lee (*Lectures on Midwifery*, 1844). "Those women are most predisposed to the disease who had had hysteria or epilepsy in early life, who have suffered from injuries of the head, or who have had violent attacks

of fever, with severe affections of the brain. Depressing passions of the mind appear to produce a predisposition to the disease. Unmarried women, who are excluded from society, and often addicted to the improper use of stimulants, are peculiarly liable to puerperal convulsions and mania. Terror and other violent mental impressions, and sometimes the pains of labor alone, are sufficient to excite convulsions. The disease occurs, not only in strong plethoric young women with their first children, in such as are of a coarse thick make, with short thick neck, but in weak, irritable, nervous females. There are some cases where irregularities of diet, especially the use of very indigestible food and stimulants, appear, without any other cause that can be discovered, to give rise to the disease. There are many cases in which the peculiar condition of the nervous system of the uterus appears to be the sole cause, and in all cases it is the principal predisposing cause, for the fits of convulsions occur in women in the first pregnancy and labor, and at no other time but during pregnancy and labor; and they often suddenly cease when the labor is completed, after every remedy has been employed without avail, except artificial delivery. *The condition of the brain on which the loss of consciousness and convulsions depend* is obviously produced by sympathy with the nervous system of the uterus; and the fits return and increase in violence till the uterus is emptied of its contents, and it is on them the irritation of the nerves of the uterus alone depends."

Dr. Fleetwood (*Theory and Practice of Midwifery*). "*It is exceedingly difficult to state anything very definite as to the cause of epileptic puerperal convulsions.* Doubtless they arise from the *sympathy of the brain* with the irritation of some different and often distant organ, it may be the stomach, the uterus, or the bowels. Intemperance in eating or drinking may give rise to it. Persons previously afflicted with convulsive affections are certainly predisposed to them at this time. Mental emotions and frights occasionally cause convulsions. In some cases, doubtless, they are owing to the efforts made during the labor-pains, by which an accumulation of blood takes place in the head. Atmospheric influence appears to have some effect in determining the frequency of the disease."

Dr. Locock (*Cyclopædia of Practical Medicine*, 1845). "The immediate causes of puerperal convulsions are often *very obscure.* They appear sometimes *to depend on a loaded state of the vessels of the brain;* at other times the brain appears to be influenced by distant irritation, either in the uterus or in the digestive organs; and again, in some cases, puerperal convulsions are induced by a peculiar irritability of the nervous system. The immediate attack may be brought on by a loaded or dis-

ordered stomach, or by food, however small in quantity, of an indigestible kind.

"Some substances, shell-fish for instance, have been found very frequently to induce convulsions in the puerperal condition, when at other times they may have been taken by the same individual with perfect impunity. A sudden fright, afflicting intelligence, or any unexpected or depressing mental emotion may excite the paroxysm. The violent straining caused by labor-pains, and even the disturbance of the frame by the earlier uterine contractions, *causing a temporary rush of blood to the head*, will sometimes bring on convulsions."

Dr. Collins (*Practical Treatise on Midwifery*, 1838), speaking of the relative frequency of convulsions in head presentations, observes, "This fact might be brought forward to support the opinion that puerperal convulsions were caused by the irritation produced in the dilatation of the mouth of the womb. This, however, is not the case, as we not unfrequently find patients attacked when the os uteri is completely dilated, and all the soft parts are relaxed. *I conceive we are quite ignorant as yet of what the cause may be, nor could I ever find on dissection any appearance to enable me to even hazard an opinion on the subject.*"

Dr. Merriman (*Synopsis of Difficult Parturition*, 1816). "There have been three especial causes assigned as usually producing this disease: 1st, General irritability of the constitution; 2d, Irritability of the uterus from distention; 3d, An overloaded state of the system. And practitioners have been influenced in their treatment of the complaint by the opinions they have entertained of its cause: thus, those who have attributed the convulsions to general irritability have considered opium as the proper remedy; those who have thought distention of the uterus the cause have recommended immediate delivery; those who believe an overloaded state of the system to be the cause of the convulsions employ large bleedings and other evacuants."

The views of Dr. Marshall Hall (*Constitutional Diseases of Females*, 1830) are also presented by the same author (Tyler Smith) as the basis of his own. The former writer says, "The principal causes of puerperal convulsion, besides the peculiar condition of the uterus itself, are indigested food, a loaded and morbid state of the bowels, a distended condition of the bladder, etc., mental shock or anxiety, muscular effort, hemorrhage," etc. Dr. Hall further believes that all these and similar causes act upon the *spinal marrow* and its system of excitor and motor nerves; and this view Dr. Tyler Smith proceeds to develop at greater length than Dr. Hall, but in accordance with his physiological and pathological doctrines. He concludes that

the convulsions are due to irritation of, or pressure upon, not the cerebrum, but on the medulla oblongata or the spinal cord; that this irritation may be either direct, or centric, or reflex. Among the centric causes operating directly on one or both of these centres he includes a toxic condition of the blood, produced by many causes, as constipation, asphyxia, etc. And among these latter he alludes to "Albuminuria, sometimes present, and which also appears to be caused by pressure on the kidneys and the renal vessels and nerves," and "accumulates noxious elements in the blood." No other allusion to the renal condition is made.

Ch. D. Meigs, M.D. (*Females and their Diseases*, Philadelphia, 1848,—"Obstetries," 1852), expresses himself decidedly against the Bright's disease and uræmic theories. He thinks that pressure of the gravid uterus upon the greater abdominal vessels produces an "excess of determination of blood to the brain," which may cause œdema and convulsions. He rarely allowed his patients to lie on the back to be confined, so as to prevent an excess of pressure. Noticeable is the following sentence: "The profound insensibility (in eclampsia) ought to be regarded as an anæsthesia caused by the presence of much black blood in the brain; when it grows blacker, so as to render the patient dark like an Ethiop, the convulsion is nearer to its close; when circulating in the cerebellum the convulsions cease, and if it pervades the medulla oblongata the patient dies from want of power in the source of the vagus nerve."

M. Bach, in an unpublished memoir written about 1850 (?), thinks that puerperal albuminuria is only *sometimes* due to albuminous nephritis.

M. Blot (*Thèse de la Faculté de Médecine de Paris*, 1849) regards albuminuria as generally unconnected with Bright's disease. He also states that he has usually met with no disease of the kidney, and insists that in the majority of cases Bright's disease has no connection with eclampsia. Of 205 pregnant women whose urine he indiscriminately examined, he found albumen in 41 only.

M. Depaul (*Leçons de Clinique Obstétricale*, pp. 293–4) also reports cases of eclampsia in which albuminuria was absent, and takes much the same ground as Blot.

Dr. Breslau (*Monatsschrift f. Geburtsheilkunde*, 1860, p. 414) reports a case of eclampsia which occurred five days after labor. After the attack albumen was found in the urine, but *not before*. After the attack ceased, the albumen disappeared. The convulsions set in with so-called uræmic symptoms. The author states that the facts are adverse to the theory of Frerichs and Braun, for no unusual quantity of urea was found in the blood examined during the attack, and no carbonate of ammonia, while

the quick cessation of the albuminuria was against the presence of heart-disease. The albuminuria, he believes, was caused by the distended condition of the lower vena cava produced by the convulsions.

Dr. Ramsbotham (*Medical Times and Gazette*, vol. i., 1863) says that of 111 cases of eclampsia 79 were primiparæ. The urine was albuminous in every five cases out of six. The author makes the following remark: "We know that fibrine is in excess in the blood of pregnant women, we have reason to believe that albumen is also in excess; and from the observations of M. Blot, as a part of that excess is got rid of by the kidneys, we may easily imagine that if there be a much greater quantity formed than healthy blood ought to contain, the superfluous albumen may be the existing cause of the convulsive seizure." He reports cases of eclampsia where no trace of albumen was present.

The late Prof. Hugh L. Hodge (*The Principles and Practice of Obstetrics*, 1864, page 99) says, "So much importance has been attached to toxicæmia during gestation as a cause of eclampsia and other nervous affections that some additional facts, illustrative of this question, must be stated; and first, therefore, let it be remembered that all pathologists acknowledge that spasms, loss of consciousness, convulsions, etc., can be produced, in the most healthy individuals, by powerful impressions on the nervous system. Such disturbances are more common in women than in men, and occur more frequently in the pregnant than in the unimpregnated condition."

Dr. Hodge argues further that this disturbance may arise just as well from a "rush of blood to the head as from a deficiency of blood, as occurs in cases of anæmia, chlorosis, exhaustion, etc. Convulsions in a large proportion of cases arise from a congestion of the blood-vessels of the brain, or from an actual effusion of serum or blood into its substance or cavities."

Dr. Hodge does not think the toxicæmic theory of eclampsia is proved. He says, "There is no essential connection between albuminuria, blood-poisoning, and convulsions."

Dr. Hodge expresses himself also against the Bright's disease theory, as Bright's disease does not exist in a great number of eclamptic patients, and where nephritis does exist, convulsions are by no means the necessary consequence. He thinks that "the presence of albumen in the urine is no positive indication of nephritis or toxicæmia in the pregnant woman. This circumstance is merely the result of renal congestion or of general plethora."

The natural exhilaration of the nervous and vascular systems and the disposition to plethora constitute, according to Dr. Hodge, the predisposing

causes of puerperal convulsions. This predisposition, he says, is therefore created by, and dependent upon, the presence of a living child in the uterus, "the action of gestation." The predisposition ceases with the death or delivery of the fœtus, gradually diminishing, and, after a few weeks, disappears. Although strictly a nervous affection, yet there is great disturbance in the circulatory system, giving origin to congestion of the brain, spinal marrow, and other organs, which will greatly aggravate the original affection, even to fatal results.

Traube (*Berliner Klinische Wochenschrift*, 1864?) does not ascribe the so-called uræmic convulsions, even in renal disease, to the retention of excrementitious matters in the blood, but believes that the loss of albumen and the consequent hydræmia cause, through the simultaneous hypertrophy of the left ventricle, a greater pressure on the arterial system, which leads to *œdema of the brain*, which shows itself as coma when the cerebrum is œdematous, and as convulsions when the middle portions are affected. The convulsions are therefore due, not to disease of the kidneys, but to a deficiency of albumen in the blood and an increased pressure on the arterial system. And this is the cause of the convulsions of Bright's disease, according to Traube.

Rosenstein (*Pathologie und Therapie der Nierenkrankheiten*, 1te aufl., Berlin, 1843, 2te aufl., 1870) avails himself of this theory of Traube's to explain the phenomena of eclampsia.

"Very much like the influence of a valvular disease of the heart upon the kidneys," he says, "acts pregnancy. By the pressure which the pregnant uterus in the last month of gestation exerts upon the vessels of the abdominal organs, especially on the renal veins, the return of the venous blood is retarded, and thereby a stagnation in the kidneys as well as in the other organs caused."

Rosenstein agrees with Virchow, who (in his *Gesammelte Abhandlungen der puerperale Zustand das Weib und die Zelle*, p. 778) makes the following statement: "Certain it is that altogether too much attention is paid to the kidneys specially, and in the liver, at least, there can be quite as frequently noticed changes as in the kidneys. In both organs (the kidneys and the liver)—and, indeed, it is a question whether the spleen too ought not to be included with them—analogous parenchymatous swellings, due to the absorption of a granular, cloudy, and apparently albuminous substance into the interior of the glandular cells, are found, in consequence of which the whole organ becomes enlarged and diminished in consistency, and after its capsule is removed appears flabby. Very often these changes present an inflammatory character, and they may then be described as

parenchymatous nephritis or hepatitis. In other cases the inflammatory nature of the change is less apparent, and we must then be content to speak of them as an albuminous infiltration. In either case the secretory function of the organs suffers, and further research is required to decide which of the two exerts the greater influence."

Regnault and Devilliers (*Arch. Gén.*, 1848), already alluded to, also held the view that the liver too undergoes changes alongside with the kidneys (calling it nutmeg-liver).

Rosenstein thinks that there must be distinguished two kinds of kidney-affections in eclampsia: 1, Such which pre-exist eclampsia, where women who suffer from Bright's disease get pregnant, pregnancy thus being a complication; and, 2, Such kidney-affections as show themselves in perfectly healthy women during pregnancy or delivery (Bright's disease produced by pregnancy). The anatomical changes of the latter are those of a stagnation hyperæmia, and have nothing to do with inflammatory processes; in cases of recovery the secretory disturbances all disappear soon after the uterus is emptied.

The *cause of eclampsia* Rosenstein attributes to some *changes in the condition of the circulation of the brain* (not to blood-poisoning), and regards co-existing kidney-disease as a favoring but *not* as a causal nor as a necessary element.

He bases his view upon the following data: 1, The uræmic phenomena are not proven to be dependent upon the retention and the decomposition of the elements of the urine; 2, There are a considerable number of cases of eclampsia with healthy kidneys and without albuminuria, or the uroscopic signs of albuminuria did not precede but followed the attack (observations by Dorn, Winkel, Brammerstädt; 3, Pregnant women having Bright's disease sometimes pass through the delivery without eclampsia; 4, The peculiar circumstances and conditions in general under which eclampsia and albuminuria occur.

According to his theory, an increased pressure such as occurs during delivery, suddenly produced in the aortic system in cases where the blood is in a high degree of hydræmia, leads to hyperæmia of the brain. But, on account of the watery condition of the blood, œdema is the necessary result of the hyperæmia. The presence of serum in the tissues, again, exerts a mechanical pressure upon the blood-vessels and produces anæmia of the brain; *and the effect of this acute cerebral anæmia is a convulsion.*

Dr. Cairns (*Edinb. Med. Journal*, Feb. 1866) reports a case where convulsions were due to shock and hemorrhage (the placenta being ruptured), and *no albumen was found in the urine.*

Dr. Putégnat (*Journal de Méd. d. Bruxelles*, 1866) reports several cases of eclampsia where *no albumen* was found in the urine. He ascribes some of the cases to mental emotion.

Dr. Sourronille (*Arch. de Tocologie*, 1875, p. 498) mentions also a case of severe eclampsia in which no trace of albumen could be detected, and no œdema.

Dr. J. Braxton Hicks, in the *Transactions of the Obstetrical Society of London* for 1867, reports four cases of puerperal convulsions on which he founds a paper on the *Pathology of Puerperal Eclampsia*. In Case 1 the convulsions came on in the seventh month; delivery took place; the attack abated, and then ceased; about the sixth day, pneumonic symptoms appeared, and she died on the tenth day. The urine before the attack was free from albumen; twelve hours after, it was highly charged with albumen, with waxy and epithelial casts. Next day the urine was diminished, was higher colored, contained albumen, blood-disks, and uric acid crystals. This continued for six or eight days, the albumen gradually disappearing before she died. There was no anasarca before the first attack, but she had some unpleasant sensations in the head for a day or two before, with partial loss of sight, etc.

Case 2 was attacked in the early part of her first labor with puerperal eclampsia repeated with great frequency, and delivery was ultimately accomplished by craniotomy; the attacks gradually subsided and the next day she became sensible. The urine drawn during the early part of the case contained not a trace of albumen; that drawn on the first and subsequent occasions after delivery contained large quantities of albumen. After cessation of lochia there was no albumen. No microscopic examination. No œdema or other symptoms beforehand.

Case 3 during application of instruments had an epileptiform fit, twice repeated after labor. Urine, after first attack, had so small a trace of albumen that it was doubtful. Nine hours after, its presence was strongly marked. Twenty-eight hours after the first attack no trace of albumen was detectable. Recovery rapid. No abnormal symptom before confinement.

Case 4, a girl of 14 years at full term, had convulsions of great severity, when the head was near the outlet. The urine drawn after the placenta was expelled contained no albumen. The fits continued, with coma between. The urine drawn off twelve hours after the first time contained copiously of albumen.

There was no *post-mortem* examination of either case which died. Yet in the paper alluded to he says, "But that in a certain proportion of

cases neither chronic Bright's disease nor any organic affection of the kidney has existed, the cases I bring forward will prove."

Quoting in connection with these cases some experiments, according to which twenty-four hours must elapse after the kidneys have ceased to act before symptoms of uræmic poisoning can occur, he says, "If this point be granted,—and it seems that, so far as our present knowledge goes, it must be,—then the only modes of explaining the occurrence of acute nephritis are in one of these three ways: either, 1st, that the convulsions themselves are the cause of the nephritis; 2d, that the convulsions and the nephritis are produced by the same cause,—*e.g.*, some detrimental ingredient circulating in the blood, irritating both cerebro-spinal system and other organs at same time; 3d, that the highly-congested state of the venous system as is produced by the spasm of the glottis in eclampsia is able to produce the kidney complication."

Winkel (*The Pathology and Treatment of Child-bed*, Rostock, 1869. Translation by Chadwick, 1876) takes a very decided view opposed to the uræmia theory. He says, "The *condition of the brain*, as revealed at the autopsies of those dying of eclampsia, is, in the first place, that of extreme anæmia with more or less marked œdema and obliteration of the convolutions; much more rarely (in only about one-sixth of the cases) there has been found extensive hyperæmia, or even capillary ecchymoses and apoplectic deposits as large as pigeons' eggs. The brain has very rarely (2–3 per cent.) appeared to be perfectly healthy. The condition of the kidneys is, on the other hand, almost the opposite, since in 35.7 per cent. these organs have been found healthy, and only in 64.4 per cent. were there detected any decided lesions (Brammerstädt). The comparative number of sound kidneys is, therefore, considerable, being more than one-third."

The urine contains as a rule (in 84 per cent.) albumen; but albumen is found sometimes also in normal and perfectly healthy pregnant women. Albumen appears often only after the attacks are over. *Thus the presence* of albumen in the urine is by no means a constant phenomenon of eclampsia; nor is there any greater uniformity in the presence of casts. Winkel agrees with Traube and Rosenstein that in most cases eclampsia is due to an affection of the brain.

Niemeyer (first American, 1870, from seventh German edition, and probably previous editions) says, "We are quite ready to admit that in most cases of *eclampsia puerperarum* there is renal disease; but we deem it quite inadmissible to attribute the albuminuria, dropsy, and eclampsia to Bright's disease."

Schroeder (*Manual of Midwifery*, English edition, 1873, from the third German) is best satisfied with the theory of Traube and Rosenstein, already given, but says also that the etiology of these convulsions is by no means sufficiently known, and that it is highly probable that the clinical picture here called eclampsia may in future be broken up into several morbid processes widely differing in an anatomical and pathological point of view.

Dr. S. Cummins (*Arch. de Tocologie*, 1874, p. 694) describes a case where labor-pains produced *general* emphysema (emphysema of lungs proper being a constant sequel of eclampsia), and argues that the emphysema causes a venous congestion all over the body and in the kidneys, too, where the efferent veins of the Malpighian bodies cannot empty themselves freely into the renal veins, and produce albuminuria, but the latter is not the cause but the *consequence of the convulsions*. He found eclampsia to occur most often in illegitimately pregnant women.

Dr. Martell (*Arch. de Tocologie*, 1876, p. 184) found only a trace of albumen in a case of eclampsia which terminated in death. On *post-mortem* examination he did not find any changes in the kidneys.

In another case reported by him (the same *Arch.*, 1877, p. 236) he found the kidneys a little congested, and slight discoloration of the cortical substance.

The late Prof. Joseph Carson, in a review on puerperal eclampsia (*Amer. Jour. Med. Sciences*, April, 1871) concludes from a careful study of the subject that puerperal eclampsia and epilepsy are analogous affections, and that neither the determination of blood to the head nor urea or carbonate of ammonia constitute the cause of convulsions. He does not admit any connection of puerperal eclampsia to kidney-disease, and considers albuminuria—which he regards as identical with hemorrhage—only as far as its relation to anæmia. Anæmia of the brain he considers to be the causal lesion of eclampsia.

Dr. Hiram Corson, of Conshohocken, Pennsylvania, who has seen very many cases of puerperal eclampsia in a large country practice extending over nearly fifty years, and who has published the cases he had prior to the year 1857 in the *Transactions of the Pennsylvania State Medical Society*, under the title *Midwifery in the Country*, writes, under date of April 3d, 1878, "I have almost no belief in albuminuria preceding convulsions. I would in every case of a pregnant woman who had the symptoms of deranged or diseased kidney, and who, from that cause, seemed threatened with convulsions, treat her, not for poison in the blood, but to relieve a deranged nervous system. The vice is in that, not in the kidney, which is only secondarily affected."

Prof. Ellerslie Wallace, of this city, in a communication to the writer dated April 23d, says, "I have seen several cases of albuminuria in pregnancy where there were dropsy, etc., and in some cases strongly expressed. Treatment, during the latter weeks of pregnancy, relieved, but did not remove the albuminuria. I was anxious about such cases, of course, and I placed such women under anæsthetics in the early part of the second stage of their labors, and delivered them by the forceps. And in not one of these cases did I see puerperal convulsions.

"I have seen puerperal convulsions plainly induced by the excessive pain of the labor, in several instances, in women who had passed through their pregnancies without any unpleasant symptoms, and in whom there *might* have been, more than ordinarily, albuminuria. But they showed, during pregnancy, no symptoms to demand investigation as to the renal function. I saw, several years ago, a few cases of puerperal convulsions from the use of ergot in first labors, where the uterine action under ergotic excitement was severe and painful."

Prof. R. A. F. Penrose, of this city, writes, April 24th, 1878, "When a woman becomes pregnant, suffering, at the time of conception, from any of the several disorganizing conditions of renal disease, grouped roughly in the generic term of 'Bright's disease,' the developments of gestation necessarily greatly aggravate the pre-existing kidney trouble, and uræmic convulsions are not only possible, but probable, the liability to them increasing as gestation advances.

"There is *not*, however, *any necessary* or *definite* relationship between so-called 'Bright's disease' and puerperal convulsions. Individuals differ so greatly in their nervous peculiarities, as well as in the effects which pregnancy occasions, that the same condition constantly produces very different results in different constitutions.

"Albuminuria, nevertheless, whether due to renal degeneration (Bright's disease), or whether the result of that hyperæmia caused by pressure, or sympathy, or by both, should in all pregnant women excite solicitude, since it indicates imperfect performance of essential renal functions, and a consequent failure in elimination that may in some constitutions lead to convulsions, and in others may develop those innumerable nervous symptoms, of greater or less gravity, which we meet with so frequently.

"While holding these views as to the dangers attending a failure in the proper performance of the functions of the kidneys,—whether this failure be owing to chronic and incurable conditions (Bright's disease), or to temporary and remediable ones,—I also *know* that there are many cases of puerperal convulsions brought on by causes entirely independent of all

kidney disturbance. The explanation of such cases is to be found in the fact, to which I have referred, relative to the great diversity of effects caused by gestation in different constitutions. Powerful emotions, alone, have in my own experience caused terrible and fatal cases of puerperal convulsions.

"To give my individual experience: I will say that in most cases of puerperal convulsions which I have seen the kidneys were in fault,—that is, there was albuminuria; but not always, or even not often, Bright's disease. In some of my cases no albumen whatever was found in the urine. A very interesting case of this kind occurred in the wards of Blockley Hospital. The urine of this woman had been examined almost daily, for some weeks before her labor, by Dr. D. F. Woods, who was conducting an extensive series of observations on the urine of gestation at the time, and was examining, almost daily, the urine of all the pregnant women in the ward (forty-six in number). This woman's urine before her labor presented an average sp. gr. of 1024; was acid; *at no time* contained albumen, but did contain uric acid and urates in excess at labor. She had as dreadful convulsions as I have ever witnessed; was comatose for several days after. Ultimately she recovered completely."

Prof. W. T. Lusk, of New York City, writes, April 20th, 1878, "I have had cases of puerperal convulsions in which careful examination beforehand proved the absence of albuminuria. I could not say in what proportion of cases such absence exists. I have met no case in which albuminuria did not make its appearance after the outbreak of convulsions."

III. *Writers who admit that puerperal eclampsia is sometimes due to Bright's disease, but who believe that it occurs as often independently of such cause.*

Scanzoni early took part in the discussion of this question, and at first (*In der Klinisch. Vortrag. über special. Path. und Ther. d. K. des weibl. Geschlechtes*, Prague, 1854) took decided ground against the view that Bright's disease is an essential lesion in most cases of puerperal eclampsia; but more recently, in the fourth edition of his *Lehrbuch der Geburtshülfe*, Wien, 1867, says, that for more than ten years he has advocated uræmic (not carbonate of ammonia) intoxication as the cause of eclampsia; although, sometimes, nervous reflex excitability plays a very important *rôle* in the causation of eclampsia.

The uroscopic phenomena are indeed seldom wanting; albumen and tube-casts are usually found, and always in direct proportion to intensity and duration of the attacks. The whole complex of symptoms in eclampsia

is such that we may conclude upon a derangement of the urine-secreting function of the kidney.

Pathologo-anatomical observations show that perfectly healthy kidneys are in this disease very rare, although very marked lesions are also infrequent, and seldom only do we meet with the third stage of Bright's disease (atrophy).

The second stage (stage of exudation), even, is seldom well pronounced, and unequally distributed through the parenchyma of the organ. Often no other tissue-changes are found than a simple hyperæmia and swelling of the kidneys. The anatomical changes, indeed, often leave us in doubt whether they had been intense enough to cause the uræmic phenomena. But it is said that the process in the kidneys being a very rapid and acute one, the severest functional disturbances result as suddenly, which is not the case in a chronic kidney-disease, where the condition is gradually developed.

"This explanation," says Scanzoni, "is very plausible; and in favor of it are also the phenomena of the acute attacks of Bright's disease following cholera, typhus, scarlatina, and which not unfrequently terminate in death by so-called uræmic phenomena, whilst at the *post-mortem* examination the kidneys do not show any other changes than those of the first or the beginning of the second stage of Bright's disease."

Another circumstance to which Scanzoni directs the attention, and which, as he says, has been heretofore overlooked, is the abnormally-elevated reflex irritability which is peculiar to the pregnant woman, and which in the presence of even a slight functional disturbance of the kidneys, and the consequent anomalies in the composition of the blood, exert such a harmful influence upon the central nervous system as under other circumstances could occur only in very far advanced parenchymatous changes of the kidneys.

The cause of the elevated reflex irritability in the pregnant woman Scanzoni considers to be *the general disturbance in the nutrition* due to abnormal condition of the blood.

The blood of pregnant women is characterized, as it is known, by an increase of water, of fibrine, and of white blood-corpuscles, and by a diminution of red blood-corpuscles and of albumen, or, in other words, it shows the peculiarity of the blood of anæmic women; and it is a known fact that nothing favors more nervous derangements and all kinds of convulsions than anæmia.

The brain of eclamptic women is found, in cases where the convulsions did not produce marked circulatory and respiratory disturbances, as a rule to be very dense, anæmic, and more or less infiltrated with serum.

Doubtless there is some connection between the imperfectly-understood disturbances of nutrition of brain-substance, and the primary disease of the parenchyma of the kidneys.

"According to the present state of our knowledge of eclampsia," further says Scanzoni, "we come nearest to the truth if we define the cause as follows: eclamptic convulsions are in the majority of cases produced by the accumulation of excrementitious matters produced by disturbed function of the kidneys; but whether here the urea, the known or some still unknown extractive matters, or the carbonate of ammonia plays an important *rôle*, must be left to future researches.

"But what explanation," he asks, "shall be given to those cases of eclampsia, becoming in recent time more and more frequent, in which clinical observation and anatomical examination exclude all possibility of a kidney-disease and uræmic intoxication?"

Some of them are not true eclampsia, but those which are can be due to elevated reflex irritability of the peripheral nerves, and to irritation of the central nervous system and of the brain. Scanzoni thinks that these play an important *rôle*, not only in these exceptional cases, but in every case of eclampsia.

There are also some cases in which the cause of eclampsia had to be ascribed to hyperæmia of the brain and of the medulla oblongata, as besides this condition no other lesion (and no kidney-changes) could be detected. As far as the influence of the convulsions upon the state of the circulation in the kidneys is concerned, Scanzoni thinks that albuminuria may be also the consequence of the convulsions, and thus cautions not to regard albuminuria in eclamptic patients as indicating invariably Bright's disease, especially in the absence of tube-casts.

In connection with the etiology of eclampsia, Scanzoni thinks pressure to be an important causative element. He does not, however, refer to pressure upon the kidneys and the renal blood-vessels, as many other authors do, but to pressure and to tearing of nerves, chiefly of the uterine nerves during the contractions of labor, by which reflex convulsions may be very easily excited.

Thus it will be seen that although at first he very distinctly announces himself in favor of Bright's disease as the cause of puerperal eclampsia, he is still unwilling to attribute all cases to it.

Dr. Barnes (*Lectures on Puerperal Fevers. Lancet*, 1865) regards albuminuria as being evidence of the kidneys being overpowered by the double strain of having to serve as emunctories for two organisms, and thinks it probable that sometimes albuminuria and uræmia are of sudden

origin, and that the convulsions follow almost immediately upon a failure of the kidneys.

Later, however, Dr. Barnes (*British Med. Journal*, 1873) says, "Convulsions in pregnancy and labor may occur *without albuminuria*. Albuminuria may exist in pregnancy, and go on to the end without necessarily inducing either convulsion or labor.

"In some cases the albuminuria existed before the pregnancy, depending upon chronic Bright's disease. Of course, the pregnancy does not mend matters. The albuminuria persists. But if I may trust the few observations to the point which I have been able to make, convulsion is less likely to ensue than in the rapidly-produced acute form. We may imagine that in chronic Bright's disease a process of accommodation takes place, whilst in acute albuminuria the nervous centres suddenly invaded by poisoned blood are unprepared for resistance."

Chailly-Honoré (*Treatise on Obstetrics*, Paris, 1867, p. 173) states that he is inclined to adopt M. Blot's view on albuminuria in pregnancy (see page 29). He says also, however, that the urine in a majority of cases is very much like that in scarlatina, where a similar congestion of the kidneys is observed. This is the character of two-thirds of the cases. Although somewhat at a loss, therefore, in which category to place him, the writer has included him in the third.

Prof. T. A. Reamy (*Transactions of the Ohio State Medical Society*, 1868, abstract in *Half-Yearly Comp. of Med. Sci.*, 1869) says, "Puerperal eclampsia and Bright's disease of the kidneys rarely ever stand in the relation of cause and effect, unless we understand simple and temporary congestion of the cortical portion of the kidneys, resulting from mechanical pressure, to constitute Bright's disease.

"True Bright's disease is sometimes found affecting women; such cases generally proving fatal, with or without convulsions. The attack is usually precipitated by the nervous state consequent upon the nature of the blood and character of its circulation reaching a point where the elements of parturition are as the torch to a magazine. There are deranged secretion and excretion in a certain proportion of cases involving the product of hæmatosis."

Naegele and Greuser (*Treatise on Obstetrics*, Paris, 1869) state that the *autopsies* in cases of death from eclampsia in general do not give any explanation of the nature of the disease. As far as the kidneys are concerned, they are either one or both in a state of hyperæmia. Sometimes they present the first or the second degree of Bright's disease (apoplectic spots and beginning of exudation in the tubules or fatty infiltrations); the third degree—that is, atrophy of the kidneys—is rare.

Dr. S. Busey, in a paper published in 1870 (*Artificial Induction of Labor in Uræmia*), is decidedly in favor of the Bright's disease theory in eclampsia. He says, "It seems to be established:

"1. That albuminuria in pregnancy is either the immediate result of pressure of the gravid uterus, or that incipient renal degeneration or precursory tendency thereto is aggravated and determined by the co-existence of pregnancy.

"2. That certain changes take place in the constitution of the blood consequent upon pregnancy or consecutive therewith, which alterations increase in intensity during the progress of gestation, and that renal disorganization, as cause or effect, may and frequently does co-exist.

"3. Even though no renal lesion exist primarily, the long-continued operation of causes which induce albuminuria in pregnancy may and frequently does result in organic renal disease; so that, in reality, it is not so much a question of the identity of albuminuria in pregnancy and Bright's disease in their visible phenomena, as it is whether they are the effects of the same proximate causes."

Concerning the blood-change theories, Dr. Busey remarks, "It matters not whether it be a blood-disease, showing itself at first simply in the excretion of albumen through the kidneys, and eventuating in organic renal disease, or primarily a nephritis; if dependent upon utero-gestation, the latter is both the exciting and predisposing cause."

Dr. Busey reports eight cases of puerperal eclampsia, seven of which he considers undoubtedly as uræmic convulsions and illustrating the different stages of the renal lesion; but in one case there was not found any albumen, either before or after the attacks, and thus he considers that the convulsions were induced by some other exceptional cause.

In this paper Dr. Busey believes "that eclampsia is but the accessory phenomenon of Bright's disease, which is the first link of a chain of morbid changes leading to it."

In a more recent paper, however (*Address on Obstetrics*, etc., read before the American Medical Association, 1876), he has evidently changed his views, and admits that numerous causes other than those growing out of Bright's disease may produce convulsions. Although he here also states that ninety of the cases of puerperal eclampsia are associated with albuminuria, and that much the larger number of the *autopsies* of women dying of puerperal convulsions exhibit renal lesions.

It is not easy to arrive at Dr. Busey's precise views on this subject from this more recent pamphlet; but he says (p. 27), "The physiological phenomena which favor cerebral congestion lend force to the once very

commonly accepted theory that puerperal convulsions were occasioned by a determination of blood to the head. This view derives important corroboration from the anatomical resemblance of the arterial cerebral circulation in women and in the cow, to which parturient apoplexy and convulsions are mainly confined."

In a letter to the writer, dated April 17th, 1878, he writes, "I have been much interested in the precise question which you propose to discuss, and at one time held very advanced views, maintaining that puerperal albuminuria was a symptom of structural changes identical with those which occurred in Bright's disease. This opinion I have modified somewhat. But to determine the ultimate relation of the two affections I have been for several years collecting the *autopsies* of those who died from puerperal eclampsia, and as yet have not found one which does not exhibit renal disease."

Strongly as these latter remarks point to the views of the writers of the first category, it seems necessary, in consideration of the matter of the second pamphlet and what precedes them in his letter to the writer, to place Dr. Busey in the third category.

Prof. Fordyce Barker (*The Puerperal Diseases*, New York, 1874), in the chapter on puerperal albuminuria (p. 68), says, "Albuminuria often exists without causing convulsions; dangerous and fatal convulsions may occur when albuminuria is wholly absent; and, still further, the nervous perturbation which causes the convulsions may also be the cause of albuminuria, or even the convulsions themselves may be the cause of albuminuria. In the present state of science, albuminuria is no longer believed to be a symptom of Bright's disease only. In fact, it is probable that, in nineteen cases out of twenty of puerperal albuminuria, the structural lesions of the kidney implied in the term Bright's disease do not exist.

"Robin, in his recent work on the fluids of the body, has demonstrated that urinary albumen has not the same composition as the albumen of the blood, and that the albumen of Bright's disease differs essentially from that occurring in temporary albuminuria of pregnancy, as can easily be shown by its chemical reactions. The albumen of the urine in Bright's disease, when brought in contact with the oxide of copper, assumes a beautiful reddish-violet color, and produces a more or less abundant flocculent black precipitate. Now, the urinary albumen of pregnancy, when Bright's disease does not exist, while it coagulates readily by heat and nitric acid, does not exhibit any such reaction with the oxide of copper. So, also, Robin has demonstrated that granular casts are not characteristic of any particular morbid state or pathological change of structure of the kidneys."

Barker also found often the urine after the convulsive attacks during labor loaded with albumen, whilst there had been no traces of albumen during pregnancy. He believes also "that we meet with convulsions, developed by emotional causes, unassociated with any anatomical lesions."

Barker directs, also, the attention to Frankenhäuser's work *On the Nerves of the Uterus*, based on careful dissections and illustrated by most beautiful plates, in which is demonstrated a direct connection between the nerves of the uterus and the renal ganglia. Frankenhäuser reasons from his discovery that the theory of pressure on the abdominal and renal vessels is highly improbable; he thinks that albuminuria and eclampsia are due to the excitation of the uterine and renal nerve plexuses. Dr. Barker adds that this theory seems to have been anticipated many years before by Dr. Tyler Smith, of London, who suggested that the albuminuria "may depend upon sympathetic irritation of the kidneys by the gravid uterus, similar with the irritation of the salivary glands, the mammæ, the thyroid, etc., and not upon mere pressure."

"Clinical observations have established the fact that the following conditions are predisposing causes of eclampsia: viz., albuminuria, hydræmia, anæmia, uræmia, and primiparity. Perhaps I should add hereditary and atmospheric influences. The latter have been alleged as a predisposing cause also by Andral, Dugès, and other French authors, and by Smellie, Denmann, Ramsbotham, Davis, and Simpson."

Dr. Barker also reports a case of puerperal eclampsia which terminated fatally, although there was not even temporary albuminuria, nor other symptoms of Bright's disease. The kidneys were examined after death by Prof. Alonzo Clark, "who pronounced them slightly congested, but in other respects perfectly healthy."

Two other cases are reported by Dr. Barker, in both of which the symptoms of Bright's disease were present, and in one of which the diagnosis was confirmed by the autopsy. In the remaining case, which recovered, there were œdema, convulsions, and albuminuria, which persisted eleven days after delivery.

In a letter to the writer bearing date April 30th, 1878, Prof. Barker writes, "I have seen quite a number of cases in which no albumen has been discovered in the urine until after the convulsions had appeared, although I have been assured that the urine had been tested several times previous to the convulsions. In my hospital practice, in thirty-four cases of convulsions no albumen could be detected in the urine after repeated examination in eight. In four of those albumen appeared after the convulsions, lasting, however, but a few days. In my private practice, in

two cases of convulsions repeated examinations failed to detect either albumen or casts; but in three both albumen and casts appeared after the convulsions."

Dr. George J. Engelmann, of St. Louis, writes, April 20th, 1878, "I do not recall a single clear case of puerperal convulsions without albuminuria, but I do know that a patient with Bright's disease with albuminuria need not necessarily have convulsions in child-bed or during labor. Convulsions may occur during the stage of dilatation without albuminuria, due to rigidity or irritation of the os, which cease upon complete dilatation. These are reflex neuroses,—hystero-neuroses, as I call them.

"I have very carefully examined the kidneys in several cases in which death resulted, and the urine contained albumen. (I still have the specimens.) As far as I remember, the kidneys were somewhat congested, but showed no structural change."

Conclusions of the Writer.

It might be anticipated that I approach hesitatingly to conclude upon a subject on which the results of observation are so various, and on which so many better qualified have thought carefully and expressed themselves so diversely. The following I have, however, reached:

First.—There are no reasons why we should exclude from the causes of convulsions in the puerperal state those which operate to produce convulsions in the non-puerperal condition. This more particularly when we admit, as I think all must, that, let the cause be what it may, the nervous centres of the pregnant woman are generally hypersensitive, and therefore more ready to respond to peripheral stimulus—irritation of any kind—than are the nerve-centres of non-pregnant women; and this may be increased by the pressures and congestions incident to labor. This being admitted, any such peripheral irritation as the pressure of a child's head upon a rigid os, like the pressure of a tooth upon a child's gum, may excite a convulsion; or the irritation of uterine nerves compressed during muscular contraction, or emotion, whether pleasurable or painful, distress, anxiety,—all *may* excite a convulsion. And it is not impossible that such a convulsion may be fatal, as it is not impossible that such a convulsion may be fatal in the non-pregnant woman. But such a result is indeed rare, and convulsions from these causes are not generally serious. They occur most often in primiparæ, where the labor has been long and painful.

But may such convulsions as these occur after delivery, when the irritation has apparently subsided? I believe they may. For, in the first place, the irritation does not necessarily cease with the termination of the

labor. The sting of the lash by no means ceases with the omission of its strokes. And, in the second place, we know that some time is often required after the operation of a peripheral irritant through a nerve upon a nerve-centre before the latter responds. It would seem as though some time must elapse before the requisite impulse can be generated in the ganglion cells to produce an explosion the resultant of which is a convulsion. Such I believe to be the nature of the convulsive attacks in most cases reported, where there is no albuminuria before the attack, and but little after it.

Second.—I think it not impossible, even, that puerperal convulsions may be caused by the congestion to which these same centres are subjected in a hard labor, as is evidenced by the red face, the protruding eyeballs, and headache, often agonizing during a pain, which the muscular effort produces. The number of these—the congestive or apoplectic cases of the oldest authors—has, however, in the admission of all, been reduced to a minimum by the subtraction from them of the cases of reflex convulsions just referred to in my first category.

Third.—Outside of these categories, in which I would include a limited number of comparatively harmless cases and a smaller number of more serious ones, I would assign the causal lesion of puerperal eclampsia to be Bright's disease of the kidneys. The Bright's disease which I believe to underlie the large majority of serious cases of puerperal convulsions may either be present at the time the woman becomes pregnant, have preceded the pregnancy, or it may be acquired during the pregnancy. In the former case, as Dr. Barnes has said (*loc. citat.*), "the pregnancy does not mend matters," and the tendency, at least, is, by reason of the pressures and congestions naturally present in pregnancy, further to interfere with the elimination of excrementitious matters, whose secretion is already embarrassed by the renal lesion, independent of any addition thereto from the pregnant state. And yet it is a fact observed by very many that such persons by no means necessarily have convulsions before, during, or after labor, especially if they are multiparæ. This is unquestionably due to the accommodation or balance which we know to be set up in different parts of an economy where another becomes gradually involved in disease. We all know how different are the results of gradual and sudden brain-lesions, —how the former may advance to an extreme degree without giving symptomatic evidence of their existence, and how serious are the consequences of even slight degrees of the latter. But we must at least admit the pregnant woman with pre-existing Bright's disease to be in the same danger of convulsions as the woman with Bright's disease who is not

pregnant, and it is not unreasonable to suppose that the danger of the former is somewhat greater than that of the latter, and in cases of primiparæ very much greater. And thus are caused some cases of puerperal convulsions. In these cases the form of Bright's disease may be any one of those to which all are subject.

In the second series,—where the disease is *acquired*,—it is almost invariably catarrhal nephritis (parenchymatous nephritis, or tubal nephritis), of which the typical example is seen in the Bright's disease concurrent or sequel to scarlet fever.

This conclusion—that most cases of puerperal convulsions are caused by Bright's disease—is justified by the fact that these cases are almost invariably accompanied by *albuminuria*, and, where a microscopic examination of the urine is made, by *tube-casts;* that a very large number are also attended by œdema; and that where they terminate fatally the autopsy generally reveals disease of the kidneys. In confirmation of this, while referring generally to the cases reported in the past pages, I desire to call your attention particularly to an analysis of the series of cases, one of the largest on record, as well as one of the most accurate,—the microscopic and chemical examinations being often made by Prof. Flint, Jr., and that of the kidneys often by Prof. Alonzo Clark, of New York,—reported by the late Prof. Elliot, in his *Obstetric Clinic* (1868), pp. 101 to 126. The list includes fifty-one cases of albuminuria and eclampsia, of which six have no exact bearing upon the subject. Eleven are cases of kidney-disease, with albuminuria, during pregnancy and parturition, not associated with convulsions, thus leaving for consideration thirty-four cases of eclampsia. In four out of these thirty-four cases eclampsia was not associated with albuminuria, nor were any tube-casts in the urine. Of the thirty cases in which the albuminuria and eclampsia co-existed, fourteen recovered and sixteen died. *Autopsies* were made only in seven cases, with the following result: Case 3. One kidney in a state of advanced Bright's disease, and the other perfectly healthy. Case 5. Advanced Bright's disease (large white kidney). Case 11. Well-marked Bright's disease. Case 13. Fatty kidney. Case 34. Kidney enlarged and congested, but not changed in structure. Case 36. Kidney large, white, congested; weight of the two, thirteen ounces, and, under the microscope, granular degeneration. Case 47. Although albuminuria, œdema, no symptoms of uræmia; there was no convulsion until the next day after labor; coma supervened immediately after the convulsion, and she died the next day, forty-two hours after delivery. At the *autopsy*, "the kidneys weighed four and a half ounces, and were healthy under the microscope." Both lateral ventricles of the

brain were filled with bloody serum; the third ventricle contained serum and a small clot; and the fourth was filled with clotted blood. The vessels of the neighborhood were examined by Prof. A. Flint, Jr., and found to be atheromatous.

The notes on the four cases of eclampsia referred to above which were not associated with albuminuria, are as follows: Case 8. Has had three miscarriages; there was œdema of the face and upper extremities; secretion of urine normal in quantity, and no albumen present; no microscopic examination of the urine was made; eclampsia; child putrid; death of the mother; no *autopsy*. Case 19. Ninth confinement; no albumen, but great quantities of urate of ammonium, some urate of sodium, and bile; eclampsia; mother and child lived. Case 30. Primipara; no albuminuria; only two convulsions; recovery. Case 39. Primipara; eclampsia after delivery; only three convulsions; no loss of consciousness; no albumen; recovery.

Of the eleven albuminous pregnant women referred to in whom no convulsions occurred, only five recovered, and six died during or after delivery. *Autopsies* were made in three only, and in *each was found advanced renal lesion*.

Now, as to Elliot's Case 47, where there were albuminuria, œdema, a physiological confinement, a convulsion the next day, followed by coma which continued until death, and the *post-mortem* revealed the *kidney weighing four and a half ounces, but healthy under the microscope*, there were, however, brain-lesions, including a small clot on one of the ventricles, and the fourth ventricle filled with clotted blood. The vessels in the neighborhood were examined by Dr. A. Flint, Jr., and found *markedly atheromatous*. It is not stated whether the kidneys singly weighed four and a half ounces or jointly; most probably the former is intended; but in either event they would seem small. *They were not examined microscopically*. The vessels of the brain were atheromatous, and death occurred by rupture of one of them. Were these not, then, cirrhotic kidneys, attending which we constantly have atheroma of the vessels of the brain and death by apoplexy?

As to the remaining four cases in which there was no albuminuria, it will be observed that in one there was a putrid child, and no examination of the urine seems to have been made after the convulsions set in, and no microscopic examination at any time; no *autopsy*. There may still have been Bright's disease, or the convulsions may have been due to the absorption of septic matter from the putrid child. The remaining three cases were mild cases, and may be placed in my first category.

Again, take the three cases of Prof. Barker, alluded to under the abstract of his views. In two of the three, Prof. Barker acknowledges Bright's disease, and in one of these the diagnosis was confirmed by a *post-mortem* examination, in which the kidneys were found very much diseased. In the third case there was no albumen in the urine drawn from the bladder after she had had one fit, and again after she had had fifteen at least, recurring at intervals of five or ten minutes, and being profoundly comatose in the intervals. Premature labor was induced, but she died after further convulsions. At the *post-mortem* examination there were two ounces of serum in the cavity and lateral ventricles of the brain, and the cerebral vessels were congested, but the kidneys were pronounced by Dr. Alonzo Clark "slightly congested, but in other respects perfectly healthy." Here, then, is a case of severity, in which there is no albuminuria, and no renal lesions are discoverable after death. These are, at most, not very numerous. They may be slightly reduced by cases of the kind included in the first category, in which, without appreciable lesion, death occurs, just as it sometimes occurs by convulsions in non-puerperal women without any discoverable lesion; and it is not impossible that this one of Prof. Barker's, which I confess is the most striking I have met, may be of this number, especially as the case was one of peculiar distress,—that of a young woman of evident refinement and education, who had wandered from her home several hundred miles distant, and, friendless in a large city, was compelled to make her bed on a door-step, whence she was taken roughly to a police-station, in which she had her first fit. Certainly, if circumstances of this kind are ever sufficient to excite convulsions, these are such. But I believe the number may be still further reduced when we remember the imperfect character of the examinations both of the urine and the kidneys. Those only of us who have had experience in hospitals know how carelessly and hastily these examinations are often made. And in the examination of the kidneys themselves the liability to error is still greater. I hold that almost never is a naked-eye examination of a kidney sufficient to justify an assertion that it is not diseased, and especially when the naked-eye examination reveals "congestion." The microscope should always be used in these cases. For this very congestion obscures more delicate changes, so as to make them undistinguishable without the microscope, and the microscope used intelligently. And, further, can we assign a limit to the mischief of a simple congestion in a kidney, especially if that congestion be suddenly induced? Finally, if it is remembered that experience often shows temporary absence of albumen in some cases of chronic Bright's disease, and that cases have even been reported with albuminuria, dropsy,

uræmia, and death after scarlet fever, and yet the *autopsy* discovers no lesion in the kidneys,—when all these matters are considered, *I cannot but think that the number of serious cases of puerperal eclampsia which cannot be attributed to some form of Bright's disease is small.*

If I am asked whether I think it impossible that a convulsion should cause albuminuria, I answer, By no means. On the other hand, we not only have every anatomical reason for supposing this possible when we recall the increased vascular pressure which must result from a convulsion, but we have also clinical evidence to this effect in the results of the examination of the urine of epilepties immediately after a fit, by Huppert (*Virchow's Archiv*, vol. lix.) and others, who found albumen in many such cases; but the peculiarity here is that the *quantity of albumen is always trifling*, unless the cases are complicated with renal disease; whereas the albuminuria of the puerperal state, at the period of convulsion at least, is very marked. But it is not impossible therefore for the milder cases of puerperal eclampsia due to reflex irritation thus to acquire a small albuminuria during the convulsions where previously none was present. Against this we should, however, guard. But the very circumstance makes it likely that a large albuminuria is due to a more decided alteration of the structure of the kidney than the mere congestive condition which is the cause of a small one.

Now as to the *cause of these renal changes* which lie at the bottom of so large a number of cases of puerperal eclampsia. How are they induced? Two views are held, by as many sets of observers, as will be recalled from the abstracts presented. One, held by Frerichs, Braun, Litzmann, Scanzoni, and others, attributes them altogether to pressure upon the emulgent veins, caused by the pressure of the pregnant uterus, producing congestion, albuminuria, and imperfect elimination of matters usually thrown off by the kidneys. This view is favored by the fact that albuminuria and eclampsia are most common in primiparæ, and in multiparæ with twin pregnancies, hydramnos, deformed pelvis, or other condition which increases the intra-abdominal pressure. On the other hand, it is objected to this view that albuminuria very early in pregnancies, some instances of which have been observed within two months after conception, cannot be thus accounted for. And it will be recollected that Bartels, in his article on the "Parenchymatous Nephritis of Pregnancy," in Ziemssen's *Cyclopædia of Medicine*, attempts to prove the anatomical impossibility of such pressure, while he claims also that congestion of this kind should be followed by interstitial nephritis instead of catarrhal nephritis, the conditions being similar to those in mitral disease of the heart, in which, from venous

pressure, the blood is backed into the kidney and liver, in both of which is found the hard kidney of an interstitial nephritis.

Those who believe in the pressure theory attribute the albuminurias of early pregnancy to pre-existing renal disease,—which is not impossible.

The opposite party ascribe the parenchymatous nephritis to an intoxication of the blood, due to the increased amount of excrementitious matter which must enter it from the retrograde metamorphosis of the tissues of the fœtus, as well as of the mother; and consider its operation to be like that of the poison of scarlatina, which similarly induces a catarrhal nephritis. The reasons for this view are well given by Dr. Peter in the lecture already quoted, and I need only refer you to them.

I think the mistake consists in the adoption of either view to the exclusion of the other. Doubtless both contribute to it in varying proportions, according as circumstances favor the operation of one or the other. Certainly the greater frequency of the eclampsia in primiparæ, and the other situations above named, can only be accounted for on the pressure theory; while the force of Bartels's argument cannot be denied.

It must not be forgotten, as stated at the outset, that the pregnant woman is liable to acquire Bright's disease from the ordinary exciting causes of renal diseases,—exposure to cold and wet, excessive eating and drinking, or the absorption of some zymotic blood-poison; or even from absorption of septic matters, possibly from a putrid fœtus or morbid material from the interior of a partially-contracted uterus. These causes are all well tabulated by Dr. Johnson in the extract from his lectures.

Now, as to the toxic agent itself in those cases of puerperal convulsions due to Bright's disease, I doubt whether it comes properly within my task to discuss it to-night. But a very few words will dispose of it. It is very true that in a few instances only has an excess of urea been demonstrated in the blood of puerperal eclamptics. It has, however, been unmistakably shown to be present in some. Frerichs, it is well known, sought to prove that it was not urea, but carbonate of ammonium into which the urea was converted, and both Prof. Hammond, of this country, and Dr. B. W. Richardson, of London, have ably refuted it, although Spiegelberg has recently reasserted the original view of Frerichs, based on a series of experiments conducted by himself and Heidenhain (London *Lancet*, 1870). Hammond and others have also proven that the urine itself is a much more efficient agent in producing the symptoms of the so-called uræmia; and there is little doubt in my mind that it is not urea, or carbonate of ammonium, or any single substance, but it is the entire mass of excrementitious

substances usually eliminated by the kidneys which, retained in the blood, give rise to the uræmic symptoms of Bright's disease, and of those cases of puerperal eclampsia depending on Bright's disease.

If it be asked how it happens, on the supposition of so grave a disease as parenchymatous nephritis, that recovery is ordinarily so rapid when labor has successfully terminated, the answer is again easy. Let it be remembered that we have ordinarily a case of catarrhal nephritis of short duration intensified by embarrassed circulation, if not primarily caused by it. This cause removed, the blood moves freely, the kidneys act rapidly, the quantity of urine is largely increased, and with it the excrementitious matter. The convalescence is therefore rapid. It is, however, no less so, and the case differs, indeed, in no way from that in catarrhal nephritis after scarlatina, where the toxic agent has not operated for so long a time or so virulently as to produce chronic changes. And it is well known that the earlier the symptoms appear, and the longer the disease has lasted before relief comes, and therefore the more deep-seated the lesions, the longer the albuminuria and the casts continue afterwards; while in a certain class of cases these symptoms continue, the relief does not come, the disease becomes chronic.

In the discussion which followed the reading of the paper, the following remarks were made:

Dr. Albert H. Smith said he fully agreed with the views expressed in the paper and the conclusions arrived at; even going further than the author, in stating that, so far as his experience had gone, he felt justified in adopting the belief that all *genuine* puerperal convulsions are the result of renal disease. He would divide the convulsive seizures arising in pregnancy into two classes,—those from hysterical and those from renal causes; the former so easily distinguished from the latter in the character of the spasmodic movements in the facial expression, and in the partial consciousness and appreciation of surrounding conditions, as to leave no question as to their true nature. Convulsions arising from brain-hyperæmia or from brain-anæmia he had never seen as attendant upon labor. In every case of true puerperal convulsions he had found great quantities of albumen, and in all cases examined microscopically casts were present.

The theory of the albumen in the urine found at the time of convulsions, and not recognized previously, being the result of the brain disturbance, was hardly tenable, when we consider the great amount of albumen found in these very cases of sudden appearance, the urine in many instances solidifying under heat, so that a test-tube or spoon in which it is heated

may be overturned without disturbing it, and in malignant and fatal cases profuse hæmaturia being present. We have all of us seen albumen in the urine detected after convulsion from brain-tumor, epilepsy, or other central conditions, but it is in quantities so small as to require delicate tests to discover it; Heller's test, for instance, giving a faint zone only at the margin of the acid.

The theory of anæmic origin, so strongly urged some years ago in a review in *Hays's Journal*, by the late Prof. Carson, is from an experimental point of view equally untenable. If the condition of anæmia bore any relation to convulsions as cause to effect, then, at any rate, we ought to have convulsions occurring after flooding; for if a slowly-developing anæmia, to which the brain would gradually accustom itself, would be a cause of eclampsia, how much more would the sudden abstraction of blood be attended with violent convulsions! But so far experience does not sustain this position that no relation between the two is ever seen in obstetric practice.

Dr. Smith had seen a vast number of cases of hemorrhage, both from placenta prævia, occurring weeks before labor, and from flooding after labor, and had seen the subjects of these conditions as nearly moribund from loss of blood as could be to live, pulseless, and with respiration almost suspended, and yet never had he seen puerperal eclampsia following such a case.

That the condition of pregnancy in itself predisposes to renal disease cannot be questioned; why or how, cannot be explained. But it is a matter of common experience that in cases of chronic albuminuria, when, under treatment, a diminution of the renal symptoms has taken place, the occurrence of pregnancy kindles up the spark into an active flame, and the chemical and microscopical tests and the physical and rational signs all show a marked and steady increase of the kidney-trouble. Dr. Smith has had a patient for over nine years with chronic Bright's disease, in whose case treatment temporarily subdues the condition; but so rapidly does pregnancy aggravate it that on two occasions the examination of the urine, showing a great increase of albumen, has enabled him to diagnosticate pregnancy before the suppression of the menses had led the patient herself to suspect it. But in addition to this tendency to albuminuria from the mere condition of the pregnant woman, we have a powerfully-exciting cause in the pressure of the gravid uterus upon the renal vessels, more apt, as Dr. Tyson has shown, to occur in primiparæ; also, as is seen, in women having a long rest between their pregnancies, in women with multiple pregnancies, and with pressure from the distention of a uterus with

amniotic dropsy. And it is in these cases in which the development of the renal symptoms is rapid that we have the greatest tendency to convulsions and the most violent and fatal form of them when they arise.

Dr. Smith agreed with Scanzoni, that the more decided the evidences of renal disease, the more violent the convulsions and the more apt to be fatal. Recovery after the appearance of marked hæmaturia, with urine solidifying under heat, is the exception, and yet in cases in which the urine becomes albuminous early in pregnancy, and continues slowly to increase in this condition, we rarely have convulsions, unless set up by a sudden aggravation of the kidney-lesion by some rapidly-developing cause. In this respect there is a resemblance to the history of the albuminuria of scarlatina, in which we observe that in those cases in which the renal trouble shows itself early, and proceeds by a steady course, we very rarely have convulsions; whereas in cases suddenly becoming œdematous, and showing the presence of large quantities of albumen, from exposure to cold, we very frequently have those symptoms culminating in eclampsia. And we have in the scarlatinal uræmia, in its complete history, a very strong argument by analogy in favor of the uræmic origin of puerperal convulsions, based upon the precise identity in the character of the convulsive movements, in the mode of onset, and the conditions of brain following each spasm, as well as those resulting from a continued repetition of them. The fact already mentioned, of convulsions having their violence and fatality proportioned so fully to the rapidity of development of the renal disease, observed as common to both affections, is further sustained as an argument by another equally well known fact, that often the first condition of things to call attention to the kidneys is the simultaneous appearance of anasarca and convulsions in patients previously considered in good health, the one in a normal physiological state of pregnancy, the other as fully convalescent from a mild attack of fever. And as a further point in the analogy, we often make in both conditions, even when anasarca and the general evidence of renal troubles are present, fruitless examinations of the urine for the discovery of either albumen or casts; finding at one time such conditions, while at others they are entirely absent and yet without any change in the general symptoms. But no one would consider this as militating against the theory of scarlatinal eclampsia being renal in its origin: why, then, should the fact that some observers have at times failed to find albumen in the urine of patients, *before* labor, who had eclampsia, followed by the *detection* of albumen during labor, be looked upon as a conclusive argument against the renal origin of puerperal convulsions?

Dr. Fred. P. Henry said that Dr. Tyson's paper had confirmed his

belief that Bright's disease is more than usually malignant when associated with the puerperal state. Under the term malignant he referred particularly to the explosive symptoms, coma and convulsions. This fact acquires a special interest when it is pointed out that there are conditions under which Bright's disease is rendered peculiarly *benign*. On a recent occasion he had endeavored to show that Bright's disease is very favorably modified as to its explosive symptoms by its association with phthisis. At that time he had seen several cases whose study had induced him to advocate this view, and since then he had seen several more. Patients with great œdema, albumen, and casts in the urine and cavities in the lungs, have retained their consciousness until almost the last moment of life, and have had no convulsions. Any one of large clinical experience must have observed this fact. Dr. Henry had endeavored to account for it by the insufficient oxidation of the blood due to the lung-disease. It is well known that Frerichs advocated the view that it is not the urea that causes the explosive symptoms, but carbonate of ammonium, a substance containing an additional atom of oxygen. Whether this be so or not, Dr. Henry is of opinion that diseases attended with insufficient oxidation of the blood, such as phthisis, modify Bright's disease in a favorable manner as regards the explosive symptoms. On the other hand, during gestation these processes of oxygenation are very active. Oxygen is required both for the mother and fœtus, and during the active interchange of gases the accumulated urea is readily converted into a more poisonous substance, whether this be carbonate of ammonium or some other.

It is customary to speak of the puerperal period as one of hydræmia, and this view was ably advocated by the late Prof. Carson; but such opinions have generally been founded upon theoretical considerations and the gross appearances of the blood. Even the microscope cannot determine absolutely the condition of the blood; a minute examination with Malassez's apparatus will, of course, give the relative richness in cells, but will not give any idea of the total amount of blood in the body, which certainly has a bearing upon the question.

In regard to the mechanical theory of pressure as a cause of Bright's disease during gestation, by impeding the flow of blood through the emulgent veins, it would require to be shown, in order to sustain it, that the forms of kidney-disease associated with diseases of the heart, impeding the return flow of blood, are identical with the forms associated with pregnancy.

Dr. C. B. Nancrede said he did not think Bright's disease, occurring in the puerperal state, could be due to pressure, since we have it coming on early in pregnancy; nor is one kidney more affected than the other. The

form of the disease is not that which is caused by obstructions, which is brown induration. The uterus cannot press upon the emulgent vein, since we have a large amount of intestines between the two. In the cases of tumors of the uterus we do not have this disease, according to most authorities; again, we have it in multiparæ. We meet with it much more frequently in twin pregnancies, even where the element of pressure is largely eliminated.

Dr. H. Lenox Hodge said that in distentions of the abdomen from ovarian tumors, albumen is sometimes found in the urine. It seems, in these cases, to be due simply to pressure. Although the pressure may be very great, and albumen present in the urine, yet convulsions do not occur. He had never known a case of convulsions due to the pressure of an ovarian tumor. Pressure does not seem enough to account for convulsions. In pregnancy there is an unusual nervous excitability, and the blood is filled with the effete products from two beings, in addition to pressure. Those who thought that in pregnancy there was no pressure on the renal veins appear to have overlooked the fact that the pressure is caused not by the direct contact of the uterus, but by the coils of intestines filled with gas and liquid, which like air-cushions transmit pressure in every direction, around every curve and in every notch.

Much light in many disorders is thrown upon their pathology by the results of the treatment adopted, and by the subsequent history of the case. In the treatment of puerperal convulsions, free venesection is recommended by the majority of authorities, although bleeding has fallen into disuse in most other disorders. The success of those who bled freely in puerperal convulsions has been very marked. Dr. Tyson alluded to the experience of Dr. Hiram Corson as being greatly in its favor. Dr. Hodge said that his father could not recall a single case of puerperal convulsions which he had lost when he had charge of it from the beginning. He bled, and bled freely. On the other hand, a very different treatment is followed in Bright's disease, and certainly very few would dare to bleed.

Then, again, as regards the subsequent history of the case. In Bright's disease there is a history of progressive danger. It generally ends in death. In the most favorable cases, if recovery takes place, there is a tedious convalescence. In puerperal convulsions the danger is limited to pregnancy, to labor, and to a little while after labor. If this period be passed, no matter how severe the convulsions have been, there is rapid improvement and rapid restoration to health. Such different results must be due to some difference in pathological condition.

Dr. Tyson said he had already referred in his concluding remarks to the two views now held; one ascribing the result to a congestion due to pressure by the gravid uterus on the renal veins, the other to a poison accumulating in the blood, similar to that of scarlatina, and acting similarly; that against the former it is urged that abdominal tumors of equal size with the gravid uterus do not cause albuminuria; that Bright's disease sometimes occurs in the earliest months of pregnancy, when it is impossible that pressure should be exerted by the gravid uterus on the renal veins. That against the latter it is urged that, except in cases where the Bright's disease pre-existed, it very seldom originates in multiparæ, but only in primiparæ, and in multiparæ with plural births where pressure is most likely to be exerted. But since, as stated by Dr. Hodge, cases do thus occasionally occur early in pregnancy, and albuminuria is sometimes found caused by the pressure of large abdominal tumors, we must, in truth, admit the occasional operation of both causes; although he believed the second was the more frequent, especially as the form of kidney-disease almost invariably found is the catarrhal, or tubal nephritis; whereas the form found in congestion of the kidney due to obstruction is always interstitial nephritis, in which the connective tissue is the seat of proliferation.

As to the prognosis of the two conditions, scarlatinal nephritis and the nephritis of pregnancy, he had also pointed out a similarity. If the poisoning is not too profound, and no lesion results from the convulsion itself, or the kidney is not permanently damaged, recovery is likely to take place in both. And as to the therapeutics being radically different in the two cases, Dr. T. was by no means certain that bleeding would not be as good a remedy in the convulsions of acute Bright's disease as in puerperal eclampsia, and, although it is never practised, the treatment which is found to be of most service under these circumstances—*active purgation, sweating*, etc.—is of the same character as bleeding, and operates in the same general way. Bleeding might be of great advantage in the convulsions of Bright's disease, and his friend Dr. Hiram Corson had already been referred to (in the body of the paper) as having used it with advantage. The difference in the two conditions lies chiefly in this, that the kidney is generally less damaged in the puerperal condition, and, having the cause removed, the organ is enabled to return rapidly to its original state and perform its function of elimination; while in the scarlatinal nephritis sometimes, although not always, the poison has operated to such a degree as to produce permanent organic change in the organ, which cannot be removed, and thus the acute condition becomes a chronic one.